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THE VISUAL MECHANISM IN DIABETES MELLITUS

(A Comparative Study of 2002 Diabetics, and 457 Non-Diabetics for Control)

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I. INTRODUCTION

THE use of insulin has wrought a striking change in the diet, the weight, and the prolongation of life of the coöperative diabetic, and at the same time it has altered the type of complications in diabetes. With insulin, Joslin[1] states that coma as a cause of death among diabetics has fallen from sixty per cent to four per cent, whereas vascular disease as a cause of death in diabetes has risen from seventeen per cent to nearly fifty per cent. Thus, through regulation of the carbohydrate metabolism and of the water and salt metabolism, insulin carries the patient through the hazards of the early stages of diabetes into a period made safer through education in a diabetic régime, and safer because of the fact that diabetes grows milder in older people.

At the outset, it must be recognized that all ocular abnormalities in diabetics need not be the result of diabetic processes, but that such abnormalities may occur quite incidentally in diabetics as a result of other causes, such as embryological abnormalities, toxins, endocrine upsets, and tissue senility. In order to make a clinical study of causes of ocular abnormalities, it has seemed to us desirable to apply searching examinations alike to diabetics and non-diabetics; to gather numbers sufficient to be of statistical value; to correlate the eye findings with the medical findings; and finally to draw deductions from a careful study of the data obtained. To carry out the purposes of this study, we have examined and wish to report the findings obtained in 2002 diabetics from the New England Deaconess Hospital, and in 457 non-diabetics from the Massachusetts General Hospital.

We are grateful to Dr. Elliott P. Joslin, who provided every facility to make this study possible, and who gave freely of his time and counsel. We are indebted to Dr. Joslin and his associates for free access to the patients and the medical records of the diabetic series; to Dr. J. Howard Means for the patients and the medical records of the non-diabetic series; to the Chemical Foundation for funds secured through the intermediation of Dr. Joslin to subsidize the work through the years 1930-33; to Dr. Edwin B. Wilson and Dr. Carl R. Doering, of the Harvard School of Public Health, for their helpful coöperation in the statistical analysis; and to Miss Dorothy Donnell, R.N., Miss Lennie Carlson, R.N., and Miss Harriet Hall, R.N., for their help with records and perimetry.

Each diabetic patient received a careful refraction, a record of the function of the extrinsic and intrinsic ocular muscles, peripheral visual fields by perimeter, blind spots and central fields by tangent screen, a study with dilated pupils of the anterior segment of each globe by slit-lamp and of each posterior segment by ophthalmoscope, and finally a Schiotz tonometer reading for each eye. Each non-diabetic patient received a pinhole vision record for each eye, and a study with dilated pupil by slit-lamp of the anterior ocular segment and by ophthalmoscope of the posterior ocular segment. The recorded medical data embraced heart size (determined by percussion and by x-ray plate), condition of peripheral vessels (determined by palpation and x-ray), blood pressures, infections, blood sugar, NPN, cholesterol, and calcium, urinary constituents, insulin, and when available the electrocardiogram, basal metabolic rate, carbon dioxide combining power, and renal function tests.

Although many of the patients received subsequent medical and ocular examinations, the findings reported in this paper include only the first medical and ocular examination, except where indicated. The recorded medical and ocular examinations were always consecutive, and represented the conditions found at one hospitalization. The presence of visual difficulties was noted in five per cent of the Joslin patients as the chief reason for attendance at the clinic. In the non-diabetic material here reported, there was not a single instance of visual difficulty as the leading symptom.

Medical and ophthalmological findings for each patient were recorded in full in three places: individual record cards, ledgers, code sheets and punched cards. The individual *record cards,* alphabetically filed, provided a ready means for follow-up of the entire eye situation, some of the patients having received a dozen

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eye examinations during the course of the work. The *ledgers*, grouping the patients by decades of age, displayed the first medical and ocular findings in tabular form, which facilitated correlation. The *punched cards*, each patient's card being derived from a code sheet and having space for 540 possible entries, provided the means for sorting and analysis of the medical and ocular data. Sorting was done at the rate of 400 cards per minute through the use of the Hollerith electric sorting machine. From the sorted material, graphs and tables were constructed to depict the incidence of ocular complications in diabetics and non-diabetics, and to bring out the relationships between ocular and general medical findings.

The most significant method of analyzing the specific findings was found to be the charting *by age of the patients*, and the charting by *duration of diabetes*. Gross incidence percentage for the entire group, diabetic or non-diabetic, may give a means for comparison with other series reported, but it fails to give a true and comprehensive picture of incidence for any complication. Charting the data *by duration of use of insulin*, and *by graded severity of diabetes* proved to be of less significance.

The 2459 cases in this series, 2002 diabetic and 457 non-diabetic, were taken as consecutive hospital admissions with no selection from an eye standpoint. Of the total cases, 297 were juveniles, under the age of twenty, and 2162 adults. The juveniles were all diabetics. For the study of purely diabetic ocular changes, unassociated with functional and organic changes which are bound to accompany increasing age, the juvenile group would seem to be the most promising. The adults, over the age of twenty, included 1705 diabetics and 457 non-diabetics of comparable ages:

Age	Diabetics	Non-Diabetics
Under 20	297	
20-39	302—17%	75—16%
40-59	776—45%	269—58%
60-up	627—36%	113—25%
	2002	457

The onset of diabetes occurred in two-thirds of the 2002 diabetics of this series after the fortieth year of life, and before the fortieth year in one-third of the total. Plates I and II bear evidence in higher blood sugar levels and greater insulin requirements that severe diabetes occurred in the young diabetics, and that diabetes became milder in older patients. The increasing age of the diabetic was attended not only by falling blood sugar levels, but also by reduced insulin dosage. Of the diabetics who could maintain satisfactory carbohydrate metabolism by diet alone without the use of insulin, there were included one per cent during the first decade of life, seventeen per cent during the fourth decade, and twenty-eight per cent during the eighth decade. The single exception to this general trend revealed by our graphs was found in the blood sugar peak of the five to ten year diabetics, plotted *by duration of diabetes*, Plate III. Insulin has been in use by the Joslin group since 1922, and without a doubt during the last ten years insulin has prolonged the lives of severe diabetics who would otherwise have succumbed. The peak of the curve above noted may be explained by the protective action of insulin, plus the slowly acquired self-education of the diabetic.

Forty per cent of our diabetics were males,

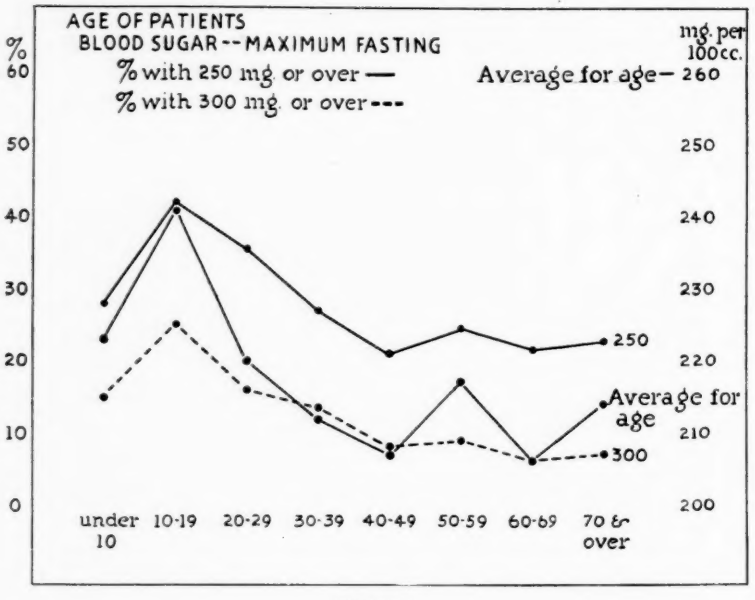

PLATE I.

and sixty per cent females. Blood cholesterol determinations were made in 280 diabetics, and of these seventy-six patients had values in excess of 229 mg. cholesterol per 100 cubic centimeters of blood. Hypertension systolic over 160 mm. Hg. was present in 486, and diastolic

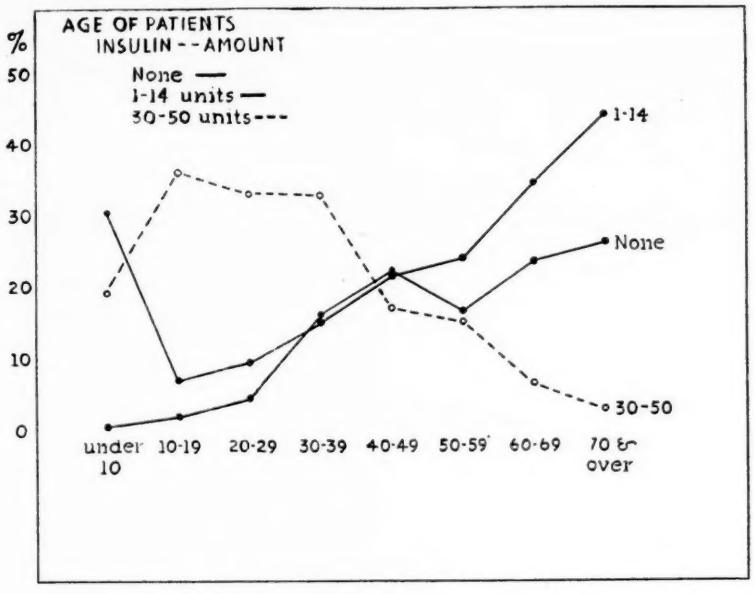

PLATE II.

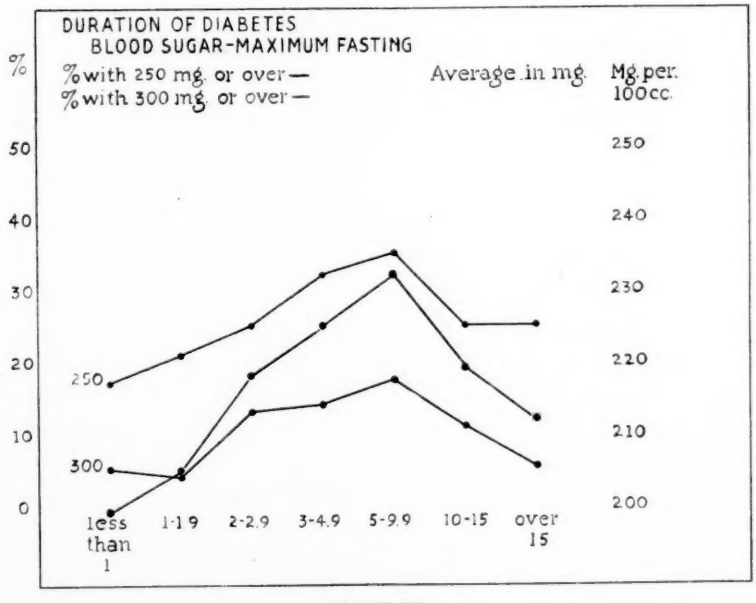

PLATE III.

showed rates of plus ten or over. Diagnoses of tuberculosis were made in fifty-four of the group, and of syphilis in thirty-three of the group.

The non-diabetics, 457 patients in all, comprised a miscellaneous group from the medical wards of the Massachusetts General Hospital. Fifty-one per cent of these patients were males, and forty-nine per cent females. Nonprotein nitrogen above 44 mg. per 100 cc. was found in twenty-seven (5.9%). Basal metabolic rates were recorded in seventy-six non-diabetics, of whom twenty-six showed rates of minus ten or less, thirty-four rates within the

over 90 mm. Hg. in 465 of the 2002 diabetic patients. Nonprotein nitrogen above 44 mg. per 100 cc. was found in seventy-six of 1583 diabetics so tested (4.8 per cent). Basal metabolic rates were recorded in 189 diabetics, of whom fifteen showed rates of minus ten or less; ninety-five showed rates within the normal range of minus ten to plus ten, and seventy-nine

range of minus ten to plus ten, and sixteen rates of plus ten or over. Diagnoses of tuberculosis were made in thirty of this group, and of syphilis in twenty-nine of the group. Of the 457 non-diabetics here included, the chief diagnoses related to the following systems:

Heart and Vessels		114
Hypertensive Heart Disease	25	
Arteriosclerotic Heart Disease	51	
Rheumatic Heart Disease	17	
Disease Peripheral Vessels	21	
Gastro-Intestinal Diseases		64
Blood Disorders		57
Bone and Joint Diseases		53
Respiratory Diseases		33
Neoplasms		32
Endocrine Disorders (not diabetic)		31
Renal Diseases		25
Diseases of the Central Nervous System		22
Liver Diseases		22
No Disease Found		4
		457

II. History of Ocular Changes in Diabetes

Changes in the eyes of diabetics have been recorded in the medical literature for nearly 150 years, the most commonly mentioned changes being those affecting the lens and the retina. In 1798, John Rollo[2], originator of the meat diet in diabetic therapy and author of the first detailed monograph on diabetic eye complications, cited the association of diabetes and cataract. This association has been amplified in numerous clinical observations during the past hundred years, and it has been given prominent place in all textbooks of ophthalmology since 1840. The more searching and discriminating slit-lamp method of studying the abnormal lens has during the past decade cast grave doubts over the existence of a truly diabetic cataract, unless it be the bilateral and fine subcapsular dot-like opacities in juvenile diabetics, first reported by Schnyder[3] in 1923, and later by Goulden[4] in 1928. O'Brien, Molsberry and Allen[5] in 1934 reported bilateral diabetic cataract of two types, found by slit-lamp in twenty patients among 126 young diabetics, aged from two years to thirty-three years, representing an incidence of sixteen per cent. Andersen[6] in 1929, using mydriasis, found no higher incidence of cataract in diabetics than in non-diabetics.

Jaeger[7] in 1855, five years after the advent of Helmholtz' ophthalmoscope, described as diabetic retinitis the combination of fine deep retinal hemorrhages and confluent waxy exudates, found most commonly at the macula, but appearing elsewhere in the retina. The presence of similar retinal changes in non-diabetic patients with angiosclerosis and vascular hypertension has led the discriminating clinician to be cautious in speaking of a retinitis pathognomonic of diabetes. The trend among recent workers having access to extensive diabetic material, such as Wagener and Wilder[8], Spalding and Curtis[9], McKee[10], Grafe[11], Gray[12], and Gresser[13], is to account for the retinitis not in terms of diabetes alone, but in terms of hypertension, arteriosclerosis, and renal impairment which may accompany diabetes.

Horner[14] in 1873 first reported the transitory refractive changes of diabetes, changes which have been thoroughly reviewed with complete bibliography by Granström[15]; Granström concurs with Duke-Elder[16] in the belief that transitory refractive changes are due only in part to paresis of accommodation, but chiefly to altered curvature and altered index of refraction of the lens itself. Elschnig[17] cited the calculation of von Hess that the aqueous would need a twenty per cent sugar concentration to account for one diopter of refractive change, and he presented the interesting occurrence of transitory refractive changes in the normal eye of a diabetic with no such changes in the aphakic eye. Thorson[18] reported the onset of myopia in a hypertensive non-diabetic following the Sippy treatment for gastric ulcer, and, curiously, the finding of two small hemorrhages in the left retina. Schieck[19] called attention to the transitory myopia which may accompany severe diarrhea, and he cited observations of others who noted transitory myopia during iritis and during icterus. Himsworth[20] emphasized the frequent occurrence of transitory refractive changes in diabetes, and pointed out that it may be the initial symptom.

Heyl[21] in 1880 reported the first case of lipemia retinalis, and the forty-second case has been added to the literature in 1931 by McKee and Rabinowitch[22]. All of these cases have been associated with high blood fat, and all of the cases have been in diabetics except one which Wagener[23] reported in leukemia following radiation of the spleen.

Krause[24] in 1903 first described the ocular hypotonus which accompanies diabetic coma, a condition which is being encountered less frequently with the declining incidence and the prompt and efficient treatment of coma.

III. Review of Important Findings in This Series

The following table shows the *principal ocular abnormalities* found in diabetics and in non-diabetics at the initial examination. In the entire paper, only the results of the initial examination are reported, unless otherwise stated in the text.

OCULAR ABNORMALITIES	4001 Eyes of Diabetics		914 Eyes of Non-Diabetics	
Wrinkles posterior cornea	1040	26%	96	10.5%
Weakness of accommodation	*165	21%		

Deep retinal hemorrhages	†730	18%	‡34	3.7%
Waxy exudates in retina	†420	10%	‡7	.8%
Depigmentation iris epithelium	258	6%	21	2.0%
Transitory refractive changes	‖246	6%		
Cataracta complicata	§246	6%	75	8.0%
Iritis	52	1.3%	12	1.3%
Atrophy of optic nerve	†27	.6%	‡4	.4%
Homonymous hemianopsia	22	.5%		
Flocculi cataract—juvenile diabetics	22	.5%		
Glaucoma	21	.5%		
Argyll Robertson pupils	20	.5%	2	.2%
Paralysis extrinsic muscles	16	.4%	1	.1%
Tobacco amblyopia	14	.3%		

*Only 759 eyes had accommodation measured. (See section "Iris—Ciliary Body.")

†Only 3893 fundi of diabetics visible. (Fundi not seen in 108 eyes because of opacities in media.)

‡Only 901 fundi of non-diabetics visible. (Fundi not seen in 13 eyes because of opacities in media.)

§Complicated cataracts in diabetics over 20 yrs.—235
" under 20 yrs.— 11
246

‖Transitory refractive changes picked up by history only in majority of cases, and incidence figure, therefore, is not complete or accurate.

IV. The Eyelids

The following eyelid abnormalities were found:

EYELID ABNORMALITIES

	4001 Eyes of Diabetics		914 Eyes of Non-Diabetics	
Squamous Blepharitis	148	3.7%	4	0.5%
Xanthelasma	59	1.4%	7	0.8%
Elongated Eyelashes	56			
Chalazion	12			
Hordeolum	7		4	
Ectropion	3			
Herpes Zoster	3			
Epithelioma	2			
Psoriasis	2			

Compared with non-diabetics there were no unusual findings in the eyelids of diabetics, and xanthoma diabeticorum was not found in a single instance. *Squamous blepharitis* in diabetics appeared 148 times or 3.7 per cent, while in non-diabetics it appeared four times or 0.5 per cent. *Xanthelasmata* were found in fifty-nine eyelids of thirty-nine diabetic patients, an incidence of 1.4 per cent, as compared with an incidence of 0.8 per cent in non-diabetics. Of thirty-nine diabetics with fifty-nine xanthelasmata, the distribution was as follows:

Sex: 34 women, 5 men.
Age: 26 patients over 50 yrs.; 13 under 50 yrs.
Side: Unilateral distribution in 19 patients.
Cholesterol: In 15 patients, values over 229 mg. per 100 cc. of blood were found in 7.

Bilateral and conspicuously *elongated eyelashes* were found in twenty-five juvenile diabetics, twenty of whom were under the age of fifteen years, and in three adult diabetics (ages 22, 30, 40). Tamaoki[25] reports that long eyelashes have been observed in undernourished children with scrofulous diseases. One is impressed with the surprisingly low number of chalazia and hordeola in diabetics of this series.

V. The Conjunctiva

The following conjunctival abnormalities were found:

CONJUNCTIVAL ABNORMALITIES

	4001 Eyes of Diabetics	914 Eyes of Non-Diabetics
Conjunctivitis	155	5
Icterus	2	17
Hemorrhage	2	3
Edema	2	0
Trachoma	1	0
Dermoid	1	0

The cases of *conjunctivitis* in diabetics were practically all of the mild type, and without complicating corneal ulceration. Bacteriological studies by smear and culture were made in forty diabetic eyes preliminary to surgery, cultures being made on Loeffler's blood serum slants, and transplants on blood agar plates. From forty conjunctivae so studied, eight yielded no organisms, and thirty-two showed organisms of the following types and frequency:

Staphylococcus albus	27
Staphylococcus aureus	8
Diphtheroids	5
B. subtilis	6
M. catarrhalis	1
M. tetragenus	1
Pneumococcus	1

VI. The Cornea

The following corneal abnormalities were found:

CORNEAL ABNORMALITIES

	4001 Eyes of Diabetics		914 Eyes of Non-Diabetics	
Arcus (see tables and and text)	367	39.8%*	179	39.0%†
Wrinkles	1040	26.0%‡	96	10.5%§
Pigment Deposits	472	11.8%	53	5.7%
"Beaten-Silver"	180	4.5%	40	4.3%
Opacities	151	3.7%	68	7.4%
Vessels	52	1.3%	11	1.2%
Pterygium	15	.3%		
Pannus	14	.3%		
Unpigmented Keratitic Precipitates	7	.1%		

*922 eyes.

†457 eyes.

‡1040 eyes:
84 unilat. = 84 patients
956 bilat. = 478 patients
1040 562 patients

§96 eyes:
4 unilat. = 4 patients
92 bilat. = 46 patients
96 50 patients

VI.—1. *Arcus*

Arcus records were omitted during the first half of our survey. Subsequently, arcus was observed in thirty-nine per cent of 922 diabetics, and in thirty-nine per cent of 457 non-diabetics, Plate IV, with parallel increase of incidence

ARCUS

Age Group	Diabetic Since 11/30/30 Patients	Arcus OD	%	%	Non-Diabetic Arcus OD	Since 11/30/30 Patients
Under 10	25	0	0			
10-19	114	0	0			
20-29	69	1	1.4	0	0	10
30-39	66	5	7.5	12.3	8	65
40-49	134	33	24.6	21.5	33	153
50-59	228	103	45.0	48.2	56	116
60-69	226	177	78.3	70.5	60	85
70 up	60	48	80.0	78.6	22	28
Total	922	367	39.8	39.1	179	457

PLATE IV.

through the various decades of each group to a maximum of eighty per cent in the eighth decade of life. Our summaries reveal no correlation between arcus and blood sugar, calcium, or phosphorus, or between arcus and the condition of the heart and blood vessels. One-third of forty-six diabetics with arcus *in whom blood cholesterol was determined* showed a cholesterol value above 229 mg. per 100 cc., as shown in the accompanying table:

46 DIABETICS WITH ARCUS

16	had cholesterol from	100-199 mg.
15		200-229
6		230-259
5		260-299
4		300-349

VI.—2. *Wrinkles*

Wrinkles involving Descemet's membrane were found in twenty-six per cent of the diabetics, and in 10.5 per cent of the non-diabetics. The wrinkles here described are invisible with the ophthalmoscope, and are seen only with the aid of the slit-lamp and corneal microscope. The larger wrinkles are seen in focal light, but the smaller ones are seen only with specular reflection and only after considerable diligence in the search. In our experience, acquaintance with the application and interpretation of mirror reflexes doubled the number of wrinkles found. The wrinkles are fine in character, vertical or oblique in direction, independent of sex, steadily increasing with age, bilateral in eighty-five per cent of the cases, central in location as a rule, variable in number but never sufficient to affect visual acuity in themselves. At no time do these wrinkles simulate the heavier ones found in hypotonus or inflammatory states.

CORNEAL WRINKLES (DESCEMET'S)

Age Group	Diabetic Patients Examined	With Wrinkles	%	%	Non-Diabetic With Wrinkles	Patients Examined
1-10	65	0				
10-19	232	1	0.4			
20-29	142	2	1.4		0	10
30-39	160	15	9.3	3.1	2	65
40-49	260	52	20.0	4.5	7	153
50-59	516	191	37.0	6.9	8	116
60-69	500	238	47.6	28.2	24	85
70 up	127	63	49.6	32.1	9	28
Total	2002	562			50	457

PLATE V.

In diabetics, wrinkles appear earlier, Plate V, they are progressively more frequent, and they are more numerous in the individual eye than in non-diabetics (numerous wrinkles in 44 per cent diabetics, and in 20 per cent non-diabetics). In diabetics, wrinkles were found in sixty per cent of the gangrene cases (47/78), in fifty-one per cent of the deep retinal hemorrhage cases (372/730), and in forty-three per cent of the diabetic neuritis cases (79/182). (The fractions used to express our findings are derived as follows: the numerator expresses the number of wrinkles found; the denominator expresses the number of cases alluded to.) If compared with the basic wrinkle incidence rate (26 per cent) these figures might seem significant, but etiologic importance is lost if correction be made for age.

BLOOD SUGAR LEVELS AND WRINKLES

Maximum Fasting	Total Patients	Patients with Wrinkles	Incidence Wrinkles
Under 140	280	54	19%
140-249 mg.	893	289	32%
250-400 mg.	409	122	30%
Over 400	24	6	25%
Unrecorded Blood Sugar	396	91	23%
	2002	562	

INSULIN AND WRINKLES

Units	Av. Age for Group	Patients	Patients with Wrinkles	Incidence
None	56	349	98	28%
1-14	54	502	187	37%
15-29	47	684	197	28%
30-49	37	375	68	18%
50 up	26	92	12	13%
		2002	562	

N. P. N. AND WRINKLES

(1584 N. P. N.'s RECORDED)

N. P. N.	Av. Age for Group	Patients	Patients with Wrinkles	Incidence
Under 35	—	878	239	27%
35-39	—	450	132	29%
40-44	—	180	75	41%
45 up	54	76	36	47%
		1584	482	

INTRAOCULAR PRESSURE AND WRINKLES

Schiotz Tension	Eyes	Eyes with Wrinkles		Incidence
Unmeasured	653	156	156/653	22%
Under 13	12	4		
13-14	21	5	126/522	24%
15-16	95	28		
17-18	394	89		
19-22	2187	584	701/2625	26%
23-25	438	117		
26-30	65	11		
31-40*	119	44	57/201	28%
Over 40*	17	2		
	4001	1040		

*Clinical glaucoma in 21 eyes; wrinkles in 8—28%.

A review of the four tables given above, attempting to correlate corneal wrinkles with blood sugar levels, insulin dosage, NPN, and ocular pressure levels fails to establish any clear-cut relationship. One is tempted to think of the process as one of tissue dehydration accompanying age, but, of course, the genesis of wrinkles must await explanation on the basis of experimental data.

VI.—3. *Pigment Deposits*

Pigment deposits upon the posterior surface of the cornea were found in diabetics over twice as frequently (11.8 per cent) as in non-diabetics (5 per cent), a probable result of the facile release of pigment from the uveal tract of the diabetic. A more complete discussion in this connection will be made in the section devoted to iris.

VI.—4. *"Beaten-Silver"*

"Beaten-silver" appearance, an irregularity of the posterior surface of Descemet's membrane and considered to be a senile change, was found with the same frequency (4 per cent) in diabetics and non-diabetics.

CORNEAE OF DIABETICS

Age of Diabetics	Beaten-Silver in Right Cornea	Beaten-Silver in Left Cornea
0-20	0	0
20-29	1	1*
30-39	2	2
40-49	5	4
50-59	33	36
60-69	35	40
70 up	9	12
Total	85	95

*Patient 28 years old.

In 180 eyes of diabetics, "beaten-silver" appearance was found chiefly after the age of fifty years, but in one patient as young as twenty-eight years. In fifty-three of the 180 eyes, pigment deposits were found in association with the "beaten-silver" appearance. Unless present to a marked degree, "beaten-silver" appearance did not interfere with visual acuity. In six eyes of three patients included in the above tabulation there was marked reduction in acuity, imbibition of the corneal epithelium, and evidence of dystrophy arising from the senile change in the corneal endothelium and Descemet's membrane, as hypothesized by Vogt[26]. Although we have observed small refractile bodies, resembling cholesterin crystals, in twenty-eight retinae and in the anterior cortex of 478 lenses of diabetics, we have never found similar bodies in the corneae of diabetics.

VII. THE IRIS AND CILIARY BODY

The following abnormalities were found in iris and ciliary body:

IRIS AND CILIARY BODY ABNORMALITIES

	4001 Eyes of Diabetics		914 Eyes of Non-Diabetics	
Paresis of Accommodation*	165/759	21.0%		
Persistent Pupillary Membrane	377	9.4%	69	7.5%
Depigmentation of Iris Pigment Epithelium	258	6.4%	21	2.0%
Acquired Pigment Deposits on Anterior Lens Capsule	112	2.8%	17	1.8%
Iritis-Cyclitis Active 14 Quiet 38	52	1.3%	12	1.3%
Pupil: Sluggish 55			26	
Dilates poorly 40			4	
Argyll Robert. 20	115	2.8%	2 32	3.5%
Horner's Syndrome	1			

*Accommodation measured in 759 eyes of patients under 50 years of age.

VII.—1. *Paresis of Accommodation*

Measured weakness of the accommodation was found in 165 of 759 eyes of diabetics, as

gauged by an arbitrary standard of accommodative power well below the accepted Donders-Duane normals. Patients over fifty years of age were not included, and, to be listed under paresis of accommodation, the eye had to be unable to attain the accommodative power listed below:

Accommodation—12 diopters	for 0-10 years of age
9	11-20
7.5	21-25
6	26-30
4.5	31-35
3	36-40
2.25	41-45
1.50	46-50

PARESES OF ACCOMMODATION
(Based upon standards above)

Age of Patient	Eyes Measured	Pareses Found	%
0-10	26	0	
10-19	207	16	7%
20-29	132	38	28%
30-39	151	46	30%
40-49	243	65	26%
	759	165	21%

It would seem that paresis of accommodation might be explainable in terms of deficient nerve impulse, reduced muscle tone, or altered character of the lens itself. In the entire series, we did not observe one case of ophthalmoplegia interna (iridoplegia or cycloplegia) such as one might expect if the accommodative weakness were wholly the result of neurogenic or myogenic causes. It is entirely possible that excessive glycogen deposition in the pigment epithelium of the ciliary body may hamper accommodation. On the other hand, the frequent association between transitory refractive change and accommodative weakness, such weakness being found in twenty-three of fifty-eight transitory refractive changes in diabetics under fifty (40 per cent), may suggest an altered lens state as a causative factor. Both of these conditions tend to disappear under a proper diabetic régime.

VII.—2. *Depigmentation*

Depigmentation of the epithelial layer of the iris was found three times as frequently in diabetics (6 per cent) as in non-diabetics (2 per cent). This proportion closely resembles the excess of pigment deposits upon the posterior surface of the corneae of diabetics (11.8 per cent) as compared with non-diabetics (5 per cent). In diabetic patients, there is frequently observed in slit-lamp study a fine radial pigment streaking on the anterior lens capsule, and in surgery a copious release of pigment to blacken the aqueous as the eye is opened. It is frequently possible to demonstrate by slit-lamp an unmistakable increase in visible pigment particles in the aqueous after massage of the diabetic eye. The 258 cases of iris depigmentation we describe were detected by transillumination, playing the slit-lamp beam upon the lens, and studying the iris in reflected light. It is difficult to explain why the uveal tract of the diabetic, particularly the elderly diabetic, releases its pigment so easily.

VII.—3. *Glycogen*

One possibility for the facile release of pigment from the uveal tract of the diabetic may be the alteration in iris pigment epithelium through selective *glycogen storage* in diabetics, demonstrated first in 1905 by Best[27], and in 1914 by Hoffman[28]. It has been repeatedly demonstrated in our material at biopsy, but only through the use of fresh tissue, fixed at once in absolute alcohol, and stained with Best's carmine. Thus we have found abnormal glycogen deposits in the retina and optic nerve, in the epithelium of the lens capsule, and especially in the pigment epithelium of iris and ciliary body, which may show an increase of many times its normal thickness due to glycogen alone.

VII.—4. *Iritis-Cyclitis*

Iritis and cyclitis were found with the same frequency in non-diabetics and diabetics (1.3 per cent). From our studies, we have been unable to recognize a distinctive "diabetic" iritis or cyclitis, and we believe that the etiology, course and end results of iritis in diabetes differ in no conspicuous way from that in non-diabetics. In our series, we did not encounter one case of rubeosis diabetica iridis (Salus). We suspect that this condition is a sequel to hemorrhagic glaucoma which is not uncommon in diabetics, rather than being a sequel to diabetes itself.

VII.—5. *Pupillary Abnormality*

Pupillary abnormalities occurred in 2.8 per cent (115/4001) of the diabetic eyes, and in 3.5 per cent (32/914) of the non-diabetic eyes. For discussion, these abnormalities may be separated into two classes: first, pupils which showed normal reactions with pronounced difficulty in mydriasis; and secondly, pupils which showed abnormal reactions.

In forty diabetic eyes, and four non-diabetic eyes, there was marked failure of normally-reacting pupils to respond to any of the solutions of mydriatic drugs. The maximum dilation obtained varied from one-quarter to one-half of the normal magnitude, and the dilation was frequently irregular, asymmetric, and unequal. According to our observations, the order of decreasing mydriatic efficiency was as follows: two per cent euphthalmine hydrochloride with two per cent cocaine hydrochloride, one per cent atropine sulphate, one half per cent scopolamine hydrobromide, and two per

cent homatropine hydrobromide. Visible synechia or other reasons for adequate explanation of poor mydriasis were lacking in all of these eyes.

In seventy-five diabetic eyes, and twenty-eight non-diabetic eyes, abnormal pupillary reactions were obtained. The majority of these eyes (fifty-five diabetic, twenty-six non-diabetic) showed sluggish reactions to all means of stimulation. Typical Argyll Robertson pupils, fixed to light, but reacting to accommodation-convergence, were found in twenty diabetic eyes, and in two non-diabetic eyes. Analysis of the ten diabetic patients presenting Argyll Robertson pupils is shown in the table, five of the patients having had lumbar puncture and spinal fluid examination.

ARGYLL ROBERTSON PUPILS

Joslin Number	Age	Sex	Blood	Spinal Fluid				Diabetes		
			Blood Wasser.	Wasser.	Protein	Cells	Gold Sol.	Duration in Yrs.	Neuritis	Hypertension
5423	52	F	3 neg.					4	no	220/110
*9478	52	M	1 neg.					2	no	
9473	57	M	1 neg.					3	no	
*3505	63	M	1 pos. (4 neg. later)					10	no	168/110
*6220	64	F	3 neg.					8	no	180/100
10892	48	M	1 neg.	1 neg.	60 mg.	0	neg.	16	yes	
10093	59	M	1 neg.	1 neg.	90 mg.	286	neg.	1	yes	
*10171	65	M	2 pos.	1 neg.	55 mg.	0	neg.	3	no	
10156	65	M	1 neg.	1 neg.	60 mg.	2	neg.	3	no	
*10759	63	M	1 neg.	1 neg.	65 mg.	2	neg.	2	yes	180/110

From the data in hand, one would strongly suspect lues in the five patients marked in the table by an asterisk: in case 9478 by history of a primary lesion; in cases 3505 and 10171 by history of primary lesion, by positive blood Wassermanns, and by supporting neurological findings; in case 6220 by one miscarriage and the loss during infancy of nine children out of ten births, and by supporting neurological findings; and in case 10759 by neurological findings. Data from the five remaining patients provide no means for incriminating lues, either in history or in clinical or laboratory findings. Three of these five patients had lumbar puncture and spinal fluid examination, with negative Wassermann in spinal fluid. The fluid in case 10093 was cloudy, contained a total protein of 90 mg., and 286 cells per cubic millimeter, probably a blood contamination. The fluid from cases 10892 and 10156 contained each 60 mg. of total protein, but no other abnormal constituents. A protein of this magnitude, while abnormal, does not indicate lues, but merely an altered permeability of capillaries.

VIII. The Lens

From this series we wish to report two types of lens abnormalities:

1. *Transitory refractive changes*, changes in the curvature and refractive index of the lens, accompanied by no visible clouding of the lens, and in the patients of our series encountered chiefly by history of abrupt blur of vision.
2. *Lens opacities* of all sorts, seen with the pupils at maximum mydriases, and with the aid of the corneal microscope and slit-lamp.

LENS ABNORMALITIES

	4001 Eyes of Diabetics		914 Eyes of Non-Diabetics	
Transitory refractive changes	246	6%	0	
*Total lenses with opacities** (one or more co-existing types)	1732†	50%	526	57%
Coronary opacities	512	12%	168	18%
Spokes in Anterior Cortex (post. 388)	728	21%	114	12%
Iridescent Crystals in Anterior Cortex	478	11%	97	10%
Fissures in Anterior Cortex (post. 199)	231	6%	39	6%
Complicated Cataracts	246	6%	75	6%

*Total lens opacities does not mean a total of opacities, but does mean a total of lenses with opacity without consideration to number or type of opacity in any one lens.

†Since the 914 eyes of the non-diabetic group all represented patients over 20 years old, we have here listed total lens opacities in diabetics over 20 years old =

3407 eyes
1732 opacities.

VIII.—A. *Transitory Refractive Changes*

Transitory refractive changes thrust themselves upon our attention by history of abrupt blur of vision in 123 diabetic patients. Fifty-

eight of the diabetic patients named above were under fifty years of age, and in twenty-three of the fifty-eight (40 per cent) a measured weakness of accommodation was demonstrated. The average magnitude of transitory refractive change was under two diopters, and the maximum change demonstrated was eight diopters. According to our observations, transitory refractive changes were not present in aphakic eyes, they were always bilateral in lens-containing eyes but not always equal in magnitude, and they tended toward myopia in the untreated patient, and toward hyperopia in the treated patient. In our patients, the highest incidence was found in the fifth decade of life (10 per cent). Undoubtedly, the incidence of transitory refractive changes would have been materially increased if search had been made by consecutive cycloplegic examinations, because changes of small magnitude would be obscured by the latent accommodation in patients under forty years of age. Studied consecutively, Himsworth[20] found transitory refractive changes in thirty-four per cent of 100 diabetics, and Granström[15] in treated diabetics found shifts toward hyperopia in fifty-seven out of eighty "fresh" cases, and seven out of thirty-eight "old" cases.

TRANSITORY REFRACTIVE CHANGES
(Tabulated by Age)

Age Group	Patients Examined	Patients with TRC	%
1-9	65	1	1%
10-19	232	13	5%
20-29	142	7	5%
30-39	160	10	6%
40-49	260	27	10%
50-59	516	44	8%
60-69	500	18	3%
70 up	127	3	2%
	2002	123	6%

Explanation of the transitory refractive change in diabetics is not to be found wholly in the sugar concentration of the ocular fluids, nor in the paresis of accommodation. Granstrom has shown that no change occurs in the corneal radius, and that no shortening or lengthening takes place in the axis of the globe, but that changes in the index of refraction of the lens nucleus does take place, changes which may be explained in terms of salt retention and osmotic interplay following precipitate blood sugar shifts.

VIII.—B. *Lens Opacities*

In the present unsatisfactory state of knowledge about the genesis of cataract, it would seem presumptuous to do more than report the number and types of *lens opacities* observable through dilated pupils with slit-lamp and corneal microscope. For this work, we used the Zeiss instrument, and 40 diameter magnification obtained with 10x ocular and A-2 objective. In reporting the data obtained, we shall whenever possible cite totals in diabetics and non-diabetics of similar ages, and we shall follow a stated scheme:

1. General incidence of lens opacities of all types.
2. Cortical changes:
   a. Anterior cortex.
   b. Posterior cortex.
   c. Complicated cataract.
   d. Flocculi in juvenile diabetics.
3. Nuclear changes:
   a. Embryonic and fetal nucleus.
   b. Zonular cataract.
   c. Adult nucleus.
4. Miscellaneous changes:
   a. Capsule.
   b. Coronary opacities.
   c. Aphakia.

Cataracts removed in capsule from the eyes of diabetics and non-diabetics were analyzed for calcium, phosphorus, and cholesterol content by Dr. Helen Updegraff Carey, who found a diminution of phosphorus in the cataractous lenses of diabetic patients, as compared with non-diabetic patients. This work is to be published[37] posthumously by Hazel M. Hunt, Director of the Chemical Laboratories at the New England Deaconess Hospital.

VIII.—B.—1. *General Incidence*

The first summary of interest is the total number of lenses in each group showing visible opacities by slit-lamp, or the other side of the picture, the total number of lenses showing no opacities by slit-lamp. Counting all types of lens opacities (fissures, spokes, wedges, equatorial opacities, lamellar dissociation, complicated cataracts, dots, vacuoles, and iridescent crystals) in diabetic patients over twenty years of age, there were 1732 lenses with one or more such opacities among 3407 eyes, an incidence of fifty per cent. The count in non-diabetics over twenty years of age was 526 lenses with one or more such opacities among 914 eyes, an incidence of fifty-seven per cent. If both groups be analyzed according to decades of age, and be graphed according to the total number of clear anterior capsules and clear anterior cortices, we have the negative information as shown in Plate VI. Diabetics through all age levels after thirty showed a greater number of clear capsules and cortices than did non-diabetics.

VIII.—B.—2. *Cortical Changes*

a. *Anterior Cortex.* The type and number of opacities found in the anterior cortex in diabetics and non-diabetics are shown in the table:

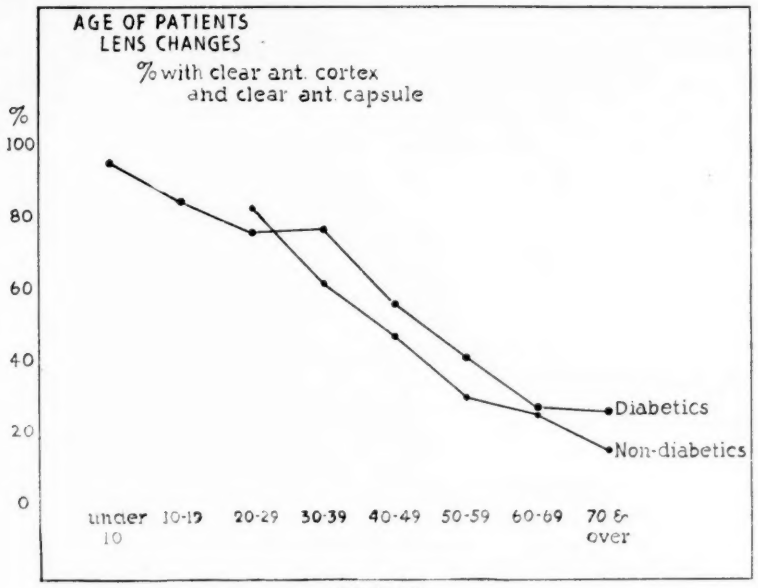

PLATE VI.

OPACITIES IN ANTERIOR CORTEX

	4001 Eyes of Diabetics	914 Eyes of Non-Diabetics
Spokes and wedges	728	114
Fissures	231	39
Lamellar dissociation	130	60
Equatorial opacities	194	49
Vacuoles	175	28
Dots	806	370
Iridescent crystals	478	97

X. *Spokes* in the anterior cortex may be studied with respect to distribution by age in both diabetics and non-diabetics, Plate VII, and those in diabetics with respect to duration of diabetes and amount of insulin.

SPOKES IN ANTERIOR CORTEX, DIABETICS AND NON-DIABETICS

DISTRIBUTION BY AGE

Diabetics Eyes Exam.	Spokes	%	Age	%	Non-Diabetics Spokes	Eyes Exam.
130	0		Under 10			
464	0		10-20			
284	7	2%	20-30		0	20
319	7	2%	30-40	1%	1	130
520	39	7%	40-50	5%	15	306
1032	220	21%	50-60	14%	34	232
999	345	34%	60-70	23%	40	170
253	110	43%	70 up	43%	24	56
4001	728				114	914

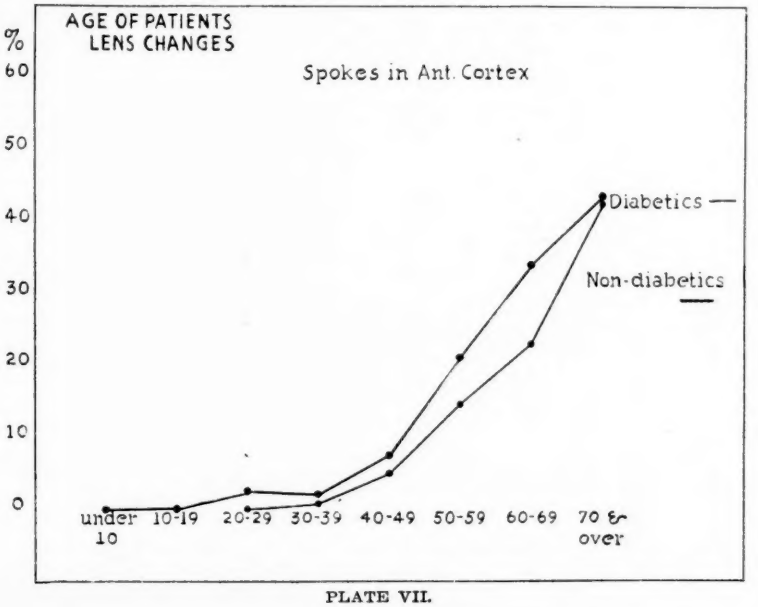

PLATE VII.

An analysis of this sort would seem to indicate that spokes are but a coefficient of age, running nearly parallel through both groups, but a little more frequent in diabetics during the fifth and sixth decades than in the non-diabetics. Spokes in the *anterior cortex of diabetics* may be studied by the duration of diabetes, and by the amount of insulin given.

SPOKES IN DIABETICS—DURATION OF DIABETES

Duration Diabetes	Av. Age Each Duration Group	Eyes Exam.	Spokes Ant. Cort.	%
Less 1 yr.	40.1	1001	119	11%
1-1.9 yr.	45.1	452	76	16%
2-2.9 yr.	45.1	378	40	10%
3-4.9 yr.	45.1	560	85	15%
5-9.9 yr.	41.2	966	203	21%
10-15 yr.	55.2	419	116	27%
Over 15	61.8	225	89	39%
		4001	728	

SPOKES IN DIABETICS—AMOUNT OF INSULIN

Amt. Units Insulin	Av. Age Each Group	Eyes Exam.	Spokes Ant. Cort.	%
None	56	697	138	19%
1-14	54	1004	248	24%
15-29	47	1366	234	17%
30-50	37	750	93	12%
50 up	26	184	15	8%
		4001	728	

If correction be made for an age factor, increasing with duration of diabetes, and decreasing with the larger doses of insulin, there would seem to be no striking relationship between spokes and the duration of diabetes or the amount of insulin administered. Neither is there any relationship of importance between spokes in the anterior cortex and abnormal blood constituents in diabetics. Of seventy-five diabetics with blood cholesterols above 229 mg. per 100 cc., spokes were found in fifteen (20%); and of 274 diabetics with fasting blood sugars above 300 mg. per 100 cc., spokes were found in forty-one (15%).

Y. *Fissures* in the anterior cortex may be studied with respect to distribution by age in both groups, Plate VIII, and in diabetics with respect to distribution by duration of diabetes and by amount of insulin.

FISSURES IN ANTERIOR CORTEX—DIABETICS AND NON-DIABETICS

(Distribution by Age)

Diabetics Eyes Exam.	Diabetics Fissures	%	Age	%	Non-Diabetics Fissures	Non-Diabetics Eyes Exam.
130	0		Under 10			
464	0		10-20			
284	0		20-30		0	20
319	2		30-40		0	130
520	12	2%	40-50	0.7%	2	306
1032	58	5%	50-60	4%	10	232
999	111	11%	60-70	7%	12	170
253	48	19%	70 up	26%	15	56
4001	231				39	914

Fissures are not so frequent in each group as are the spokes, but they seem to follow age in trend.

Fissures in the anterior cortex of diabetics:

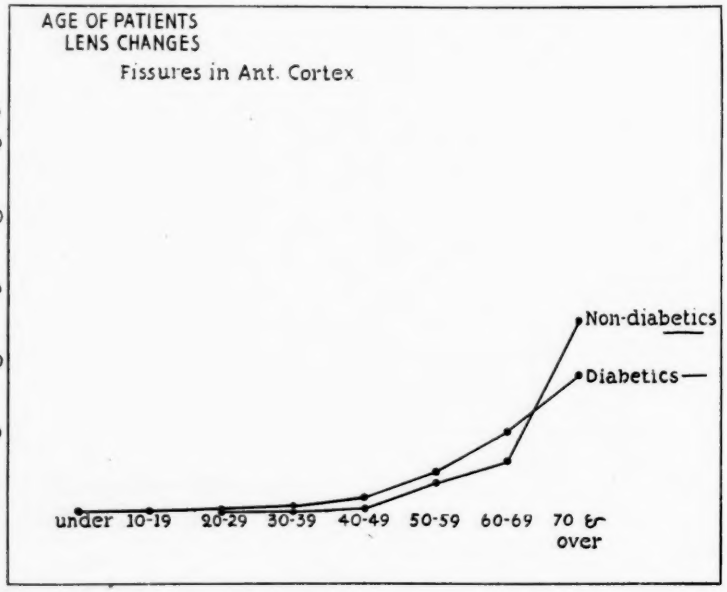

PLATE VIII.

FISSURES IN DIABETICS—DURATION OF DIABETES

Duration Diabetes	Av. Age Each Duration Group	Eyes Exam.	Fissures Ant. Cort.	%
Less 1 yr.	40.1	1001	39	3%
1-1.9 yr.	45.1	452	14	3%
2-2.9 yr.	45.1	378	10	2%
3-4.9 yr.	45.1	560	27	4%
5-9.9 yr.	41.2	966	47	5%
10-15 yr.	55.2	419	41	9%
Over 15	61.8	225	53	23%
		4001	231	

FISSURES IN DIABETICS—AMOUNT OF INSULIN

Amt. Units Insulin	Av. Age Each Group	Eyes Exam.	Fissures Ant. Cort.	%
None	56	697	53	7%
1-14	54	1004	75	7%
15-29	47	1366	73	5%
30-50	37	750	26	3%
50 up	26	184	4	2%
		4001	231	

The same remarks apply here that were given for spokes. Of 75 diabetics with blood cholesterol above 229 mg. per 100 cc., fissures were found in three (4%); and of 274 diabetics with fasting blood sugar above 300 mg. per 100 cc., fissures were found in eight (2%).

b. *Posterior cortex.* The type and number of opacities found in the posterior cortex of diabetics and non-diabetics are shown in the table:

	4001 Eyes of Diabetics		914 Eyes of Non-Diabetics	
Spokes and Wedges	378		40	
Fissures	199		23	
Lamellar dissociation	19		18	
Vacuoles	362		36	
Dots	278		50	
Iridescent crystals	316		39	
Cataracta complicata	246	6%	75	8%

Thus, the posterior cortex apparently contains relatively less spokes, fissures, lamellar dissociation, and relatively more vacuoles than does the anterior cortex. Distribution of opacities in the posterior cortex analyzed by age, by duration of diabetes, and by amount of insulin given differs in no essential way from the distribution shown in the anterior cortex.

All references will appear at the end of this paper, the second part of which is scheduled for next week's issue.

PULMONARY TUBERCULOSIS AND PREGNANCY

BY CLEAVELAND FLOYD, M.D.*

EFFECT OF PREGNANCY ON TUBERCULOSIS

THERE has been great difference of opinion as to the effect of pregnancy upon pulmonary tuberculosis. Up to eighty years ago, pregnancy, so far from being regarded as a complication of tuberculosis, was considered to be rather beneficial. (We recall the advice of Hippocrates that consumptive girls should marry.) It was Grisolle who, in the middle of the last century, first insisted on the unfavorable effects of pregnancy on pulmonary tuberculosis. Whereas the bad effects of delivery and the puerperium had been remarked, the deleterious influence of pregnancy itself was more difficult to establish, as it was masked behind a seeming improvement or arrest of the tuberculous process frequently noticed after the first months of pregnancy, and which we are now inclined to attribute to a sort of partial bilateral pulmonary compression, induced by the characteristic raising of the diaphragm, coincident with the advance of gestation.

It was gradually recognized that the whole course of a tuberculous process and the whole course of a pregnancy could not be regarded as units in evaluating the effect of pregnancy on tuberculosis of the lungs. The advancing periods of the pregnancy complicate to a varying extent the different stages of the pulmonary tuberculosis. It was also realized that old, healed or dormant lesions were not of much importance in this connection. Hence it was desirable to differentiate. This was extensively undertaken, with the result that, according to the figures of one authority (Frischbier) pregnancy induces an aggravation in the tuberculous condition in 70 per cent of the cases, but according to another (Scherer), in only 6.4 per cent. Such a great variation in results can only be explained on the assumption that the different investigators are not dealing with the same material, or are not handling it in the same way.

It is of the utmost importance to consider the fundamental situation underlying the available data. Whether one agrees that about 5 per cent of all cases of pregnancy are complicated by tuberculosis of the lungs, and that of these about one-fifth, or only 1 per cent are active, it will probably be conceded that the figures gathered from the actual experience of any one man are not in themselves conclusive. The remedy for this situation is to take into consideration the figures of various research workers, institutions

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and practitioners. Here, however, an as yet unsurmounted obstacle is encountered. There are several variables in the equation, which are now generally recognized, chief among which are the type of the infection, the stage of its progress, and the stage of the pregnancy. There has been an attempt to make allowance for these. There is a much larger number, however, rectification for which is generally not even attempted, such as the constitutional, social, economic, and mental factors conditioning the individual patient. Not only this, but the method of dealing with the figures differs so that they involve a heterogeneity even when submitted to a single man for ultimate analysis.

To illustrate: It is fair to assume that pulmonary tuberculosis will be uniformly differentiated from that specifically affecting other parts of the body, but two general types are usually distinguished in the pulmonary form, which are variously designated as progressive and stationary, active and inactive, open and closed, exudative and proliferated, manifest and latent, acute and chronic, and either of these may be unilateral or bilateral. These designations give no basis for an exact comparison. It is obvious, for instance, that a case classified as "stationary" by one reporter might quite well be in the "active" category of another, *etc.*, and yet for each, respectively, it is the second, or more serious, type which alone is usually involved in discussions of the effect of pregnancy upon tuberculosis of the lungs.

Frequently in the literature the patients are reported to be in the "first" or "second" stage of pulmonary tuberculosis, without any indication being given as to how these stages are determined. Usually, however, the three stages as outlined by Turban and modified by Gerhardt are referred to:

I Stage—Slight involvement, limited to small area of one lobe, which in bilateral cases may extend from the apex not lower than the spine of the scapula, and the clavicle; in unilateral cases, from the apex not below the second rib in front.

II Stage—Slight involvement, more extensive than I, but at most implicating half a lobe.

III Stage—All involvements more extensive than I and II, and all with any considerable cavities.

This division is based primarily upon the *extent* of the involvement, and leaves out of consideration the dynamic virulence of the infection. Other divisions, such as those of Bard-Piery-Neumann have also been used, but no schematic arrangement can fairly reflect the myriad phases and combinations in the progress of pulmonary tuberculosis.

The pregnancy itself may be divided merely into the "first" or "second" half. If a finer division is made, it is apt to be fairly uniformly into three stages, the first comprising the period from conception to sixteen weeks, the second, from the sixteenth to the twenty-eighth week of gestation, and the third, from the twenty-eighth week on, when the fetus is presumed to be viable. This seems clear and logical enough, but translated into terms of months, confusion arises from the fact that many writers tacitly imply a ten-lunar-month duration of pregnancy, whereas others speak in the tradition of nine months. A further objective source of inconsistency lies in the varying lengths of time after parturition during which aggravation of pulmonary tuberculosis is regarded as attributable to the child-bearing. Some authorities report negative effects if no signs appear within two weeks, whereas others include in their figures the results of medical observation extending for as much as fifteen years!

Aside from these variables, for which rectification has been attempted, however inconsistent with each other, there remain the variables which are rarely if ever given scientific consideration, and to which only brief allusion can be made here. It was Libermeister who said that the effect of pregnancy on pulmonary tuberculosis depends essentially upon the capacity of the sick organism. A tuberculous woman in a sound social and economic situation, eager to have the child, and able to be closely tended and cared for, will bear pregnancy better than one *with the same clinical findings*, but burdened with economic worry regarding the expense of this addition to the family, and who, so far from being able to indulge in increased rest, must perhaps work longer hours to meet this new item in the budget. For such a woman, who has just barely held her own, pregnancy may be the last straw. Then, too, a much neglected issue is that of individual type: Those with long, thin, relatively undeveloped torso seem to be those predisposed to tuberculosis, and those with whom its evolution is rapid. The stocky, thick-set type resists it better. It is quite possible that considerations of age, race, occupation and climate should also be taken into account.

If such are the obvious inconsistencies in the choice of the case material itself, no less important are the vagaries of the different analyzers in their interpretation thereof. Thus one worker, when asked for a report on the unfavorable effects of pregnancy on the course of pulmonary tuberculosis observed in the institution with which he was connected, rejected as evidence all the cases which were first examined in advanced pregnancy, for despite the fact that the inroads of the tuberculosis were great, it was impossible to establish—clinically—that the lung condition had been aggravated by the

pregnancy. Another worker, faced with a similar situation, might accept the impression of the patient as to whether the inception of the tuberculous process or its aggravation occurred in connection with the pregnancy. One might go on multiplying such instances of irreconcilability, but enough has been said to indicate that what with the diverse classifications of the disease, and of its stages, the various divisions of the period of pregnancy, the unrecognized factors of social and economic import, taken in conjunction with the divergent points of view of those who interpret the figures, the number of varying combinations possible in any apparently well-organized group of statistics would run into the thousands.

Yet even the statisticians seem to agree in general upon an aggravating influence of pregnancy on pulmonary tuberculosis. This is but natural, for the modern therapy for tuberculosis, as to both men and women, involves rest, freedom from overexertion, pain or anxiety. The aim is to produce a margin of strength by which the healing process may be furthered. All extraordinary burdens of whatever sort are definitely contraindicated, and any concomitant disease is a cause for apprehension. In this connection it is not without interest to review some of the physiological changes incident to normal pregnancy, which in any other circumstances would be considered pathological. Pregnancy makes excessive demands of calcium, iron and iodine metabolism. There is a pronounced hypertrophy of the parathyroids, reflecting the drain for calcium. A pregnant woman's basal metabolic rate during the later months is 30 per cent higher than that of a non-pregnant woman. There is a tendency to glycogen deficiency in the liver, which is most marked in early weeks of pregnancy. The renal threshold for sugar is lowered, with a resulting glycosuria. The capacity of the kidney declines progressively during the last twelve weeks of pregnancy, with the result that every woman definitely drops toward the toxemic level of urine volume during the latter part of gestation. As to the blood, there is a diminution of red corpuscles and an augmentation of fibrin and of water, with about normal albumin. Thus there results a marked relative preponderance of albumin over red corpuscles. An encroachment on the cardiac reserve is always demonstrable in healthy pregnancy. The entire circulation is upset, the pulse is quickened, the blood pressure is raised. The physical changes induced by pregnancy are calculated not only to stimulate metabolic activity, tax glandular functions and burden the eliminative mechanism, but to increase the rate of respiration, which is especially significant in connection with pulmonary tuberculosis, where rest for the diseased lung is of prime importance.

Any consideration of the effects of pregnancy upon a tuberculous woman must also take into account its climax and end results. Uninterrupted pregnancy leads to parturition, which involves severe demands on the human organism. The term "labor" is not without significance. In addition to the normal exertion involved in delivery, there is always the possibility of emergencies more serious for the tuberculous woman than for the average mother, such as infection, or the resort to anesthesia. One physiological factor in parturition which is specifically injurious to the diseased lung is the sudden decompression consequent upon the descent of the diaphragm at delivery. Not only must the complications of pregnancy and parturition be taken into consideration, but those presented by the life of the infant as well. Of course the mother should not nurse her child, but aside from this the necessary ministrations are many and insistent, involving broken sleep, lack of rest, and nervous exhaustion. If the home conditions are not such that these burdens can be spared the tuberculous mother, there is no doubt but that they must be added to the undesirable effects of childbearing upon pulmonary tuberculosis.

TREATMENT OF A TUBERCULOUS WOMAN DURING PREGNANCY

In dealing with a pregnant woman suffering from pulmonary tuberculosis, two courses are open to the physician. He may address himself primarily to the treatment of the lung condition, modifying the usual therapeutic measures only in so far as the changing physical condition of the woman requires, or he may resort to prophylactic measures aiming to protect the phthisic patient from the ordeal of parturition and the dangers of the puerperium. The first course suggests either the traditional sanatorium care, or the induction of artificial pneumothorax; the second involves abortion.

Individual variations in resistance, the extent of the pulmonary area involved, the severity of the infection, as well as the influence exerted by age, sex and race are elements in the determination of the course of the disease, to which pregnancy adds one more factor, due to its tendency to modify the behavior of the lung lesion. All these considerations must be weighed in making a choice of procedure.

Pulmonary tuberculosis can be most effectively treated in a sanatorium. The benefits accruing from an established routine of bodily rest, freedom from domestic responsibilities, and a diet relevant to the disease are in themselves notable, and may enable a woman successfully to withstand the aggravation of the tuberculous condition so frequently a concomitant of pregnancy. However, whereas sanatorium care is even more urgently indicated for the pregnant consumptive than for the ordinary phthisic, to

produce satisfactory results, it should cover the whole duration of the gestation, parturition, and puerperium, and a period of at least six months thereafter. This last is of the utmost importance, because it is the time recognized as fraught with the greatest danger of the breaking down of a healed process or the quickening of an active lesion. If the tuberculous condition is induced by the pregnancy, the sanatorium treatment cannot of course be begun until its confirmation, but the activation of pulmonary tuberculosis by pregnancy is rarely delayed many weeks, and progress has been made in its early diagnosis.

If, for whatever reason, sanatorium care is ineffective to check the progress of the disease with the advance of pregnancy, or if its benefits are not available because of the economic situation, other means of aid must be resorted to.

PNEUMOTHORAX

Artificial pneumothorax results in functional lung inactivity, by the creation of a positive intrapleural pressure which reacts directly against the normal inflation of the lung during respiration, and impedes the movement of the thoracic muscles. The effectiveness of this method in controlling pulmonary tuberculosis is well established. When a complete collapse of the diseased lung is possible, suppression of the symptoms of active disease, such as fever, cough, and expectoration, may be counted on. Even in cases where the tuberculous process has advanced to the stage of cavity formation, pneumothorax will often give striking results. The cavities may be closed, and, as a consequence, positive sputum may be eliminated, and this constant source of danger be done away with. Collapse therapy is one of the most effective in dealing with pulmonary bleeding. The acute extension of the tuberculous process as a result of aspirated blood, with ensuing tuberculous pneumonia, is thereby prevented. To insure satisfactory results, the compression of the lung, maintained by periodic refills, should be continued for from two to five years, as it is impossible to renew the treatment by collapsing a lung that has once been permitted to reinflate.

The enduring effect of pneumothorax is such that in those instances where phthisis has been apparently healed by this means before the inception of pregnancy, the latter runs a normal course without deleterious reaction on the lungs. It is wise, however, to insist upon an interval of two years between the healing and subsequent pregnancy.

Resort to pneumothorax in conjunction with pregnancy is fraught with no greater likelihood of complications than where pulmonary tuberculosis alone is the issue. Where pneumothorax has once been successfully established, it should not be discontinued on account of an intervening pregnancy and puerperium. Certain precautions should be observed, however. The raising of the diaphragm in pregnancy brings about a dislocation of the lungs. There is a reduction of the vertical diameter of the thoracic cavity, with a corresponding increase in the transverse diameter. Normally, the diminished action of the diaphragm, which this implies, is compensated for by enhanced activity of the muscles of the thorax. In pregnant women under pneumothorax, this is increased, for one lung is almost wholly immobilized by collapse. Hence, unless there be a reduction, or even a temporary cessation of refills, during the last months of pregnancy, regulated according to the condition of the patient and to the degree of her shortness of breath, an enormous surcharge of work is put upon the well, breathing lung, already embarrassed by the raising of the diaphragm, which favors making acute any inactive or initial processes. Disturbances of the circulation may also be provoked, because of the fact that the mediastinum, and with it the very important organs it contains, are subjected to a triple pressure: from the bottom up by the increased endo-abdominal pressure, from one side by the pressure exerted by the pneumothorax, and from the other side by the breathing lung which is in a state of compensatory emphysema. Hence the routine technic in producing pulmonary compression in connection with pregnancy requires mature judgment on the part of the operator that he may correctly gauge the progress of the disease, the proper interval between refills and the amount of intrathoracic pressure necessary to be maintained without causing embarrassment to the abdominal organs and the already burdened heart.

The effectiveness of this procedure depends in large part upon the duration of its application prior to the inception of pregnancy. If the collapse has not yet reached its maximum therapeutic value, whether by being begun too recently, or by reason of pleural adhesions preventing complete collapse, or by other complications, the woman is not adequately defended against the strain of pregnancy, and feels its consequences in her weakest spot, the lung. In such cases, pneumothorax may be inadequate to impede the advance of the lesions. These considerations hold with equal or greater force when the pneumothorax is instituted after the beginning of the pregnancy, or after parturition, yet even in these cases, remarkably satisfactory results may be obtained.

Simultaneous bilateral pneumothorax in pregnant women is of such difficulty and clinical delicacy that it is not to be advised except in special cases. Nevertheless, where the bilateral treatment, previously instituted, has had to be

abandoned after a relatively short period, the tuberculous process may have been sufficiently checked to have benefited the patient. In the treatment of a bilateral process under these circumstances, phrenicotomy, or, for temporary effect, nerve crushing in place of excision, may be resorted to in conjunction with pneumothorax. One lung is thus subjected to limited activity and increased intrapleural pressure by diaphragmatic paralysis, while the second can be simultaneously treated with pneumothorax. The more radical procedure of thoracoplasty will rarely if ever be employed in the case of a pregnant woman. Where it is otherwise desirable, it may prove of great value if deferred until after parturition.

ABORTION*

With the opening of the twentieth century, the dangers of pregnancy, parturition and the puerperium for a woman suffering from tuberculosis of the lungs were so firmly established that it was generally recognized that abortion should be considered as a prophylactic measure. A decade later, this practice had become the rule, and hundreds of articles appeared, particularly from the pens of methodically minded German authorities, advocating early abortion in every case of the concurrence of pregnancy with pulmonary tuberculosis, *as a matter of principle*, irrespective of the clinical prognosis of the individual case. This position was assailed not only by those who discountenanced abortion on religious or political grounds, but by physicians who questioned the necessity or efficacy of the procedure. Each side resorted to statistics to support its contention, but met with familiar difficulties. The quantitative basis of the schemes in use for the general analysis of the course of pulmonary tuberculosis rendered them peculiarly ill-adapted to serve as indices for resort to abortion, where the *rate of degeneration* is the crux of the issue.

Another serious source of error in connection with figures showing the effects of abortion on the course of pulmonary tuberculosis was that the term was sometimes restricted to technical abortion, sometimes used to cover inducement of miscarriage and of premature delivery as well. Moreover, among its effects are frequently included those consequent upon sterilization by a major surgical operation, which is a far cry from a simple manipulation under a local anesthetic.

No hard and fast rule can be laid down, a priori, as to when one should and when one should not resort to abortion, nor can the exact limits of an unfavorable prognosis in the absence of abortion be established. Certain indications may be suggested, however, based upon a thorough knowledge of the social and medical background of the patient as well as the state and course of the disease.

In the case of a healed lesion, or of an obsolete process, where the x-ray shows fibrosis or calcification, sanatorium methods of care followed in the home are all that are required to carry the patient successfully through gestation and parturition. In the latent case, where there is a history of exposure to phthisis, a few positive symptoms, no signs of physical activity and uncertain or early x-ray changes, there is no need to consider interrupting the pregnancy, if the patient can be cared for in a sanatorium, with or without artificial pneumothorax.

In the early active case of pulmonary tuberculosis, where cough, fever, moist râles and x-ray shadows, soft and flocculent in aspect, are present, pregnancy may be allowed to continue to term, provided sanatorium methods of treatment are adopted, together with the induction of pneumothorax, either at the time of the discovery of the disease, or later on in the pregnancy, if the process of infiltration advances, even deferring it until after confinement, if all the pulmonary indications are favorable.

Where pregnancy occurs in a unilateral case, already well established, as shown by signs of infiltration and moist râles involving the upper third of the chest, and accompanied by cough, expectoration, loss of weight, and positive sputum, pneumothorax should be instituted at once, if the pregnancy is to be allowed to proceed without interruption. If for whatever reason, pneumothorax is impossible, abortion should be resorted to. This should be done as soon as possible, even if from the point of view of the clinician and the roentgenologist, the tuberculous process appears in a favorable light, because in the later months of pregnancy the tuberculous deterioration is greatly accelerated, whereas the earlier the abortion is done, the greater is the assurance of success.

In those cases, however, where the disease when first detected is already in an advanced stage, and the prognosis for the mother is bad, interruption of the pregnancy would contribute little toward saving the life of the mother, and that of the child would be sacrificed. It is better, therefore, in the advanced case to allow pregnancy to proceed without interruption.

Acute miliary tuberculosis in the majority of cases leads rapidly to a fatal termination. Occasionally in a patient of relatively high resistance, the chronic form of this condition is encountered, but these cases are unusual. In neither of these situations is anything to be gained by the use of pneumothorax or by the interruption of the pregnancy.

*For the purposes of this paper, the term "abortion" is to be understood to cover all interruption of pregnancy up to the seventh month, at which time the fetus is presumed to be viable. In the legal vernacular, the term "miscarriage" seems to be preferred, but whatever the word, the idea involves an interference which renders impossible the issue of a living child.

Tuberculous enteritis, as well as tuberculous ulceration of the larynx, is commonly associated with advanced pulmonary tuberculosis, and in the great majority of cases, the prognosis is unfavorable. Where pregnancy is complicated with these manifestations of advanced disease, the duty of the physician is primarily to the child. Rarely, if ever, will interruption of the pregnancy be seriously considered in such cases.

The stage of gestation at which the interruption is undertaken is of great import to the patient. The termination of pregnancy in its early weeks is a much more simple matter than later on. Following the completion of the first sixteen weeks, on account of the development of the fetus, the increasing size of the placenta and uterus, abortion must be regarded as a major operation for the tuberculous.

The methods of producing abortion vary from the simple use of x-ray to the serious operation of hysterectomy. The experience of numerous men with the use of x-ray in the production of abortion merits careful consideration. The chief advantages of this method are its simplicity of application, its comparative innocuousness to the patient, and the fact that the resulting abortion occurs spontaneously and without uterine hemorrhage. If desired, the x-ray exposure may be so regulated that menstruation is not interfered with. Although the expulsion of the fetus may not take place for over a month after radiation, there seems to be no evidence of infection as a result of this delay.

There has been discussion in recent German literature in regard to the use of intra-uterine applications. Several pastes have been employed and have had an extensive vogue. They induce expulsion of the fetus within twenty-four hours. Occasionally the placenta requires mechanical removal. This method of inducing abortion has not received general acceptance, however.

Another procedure is that of spraying or injecting a strong solution of sodium and calcium chloride between the membranes and the uterine wall. In twenty-four hours, due to the irritating effect of the solution, abortion takes place without mechanical aid. The method is not always successful and occasionally the placenta does not come away without instrumental removal.

In surgical interruption of pregnancy, two factors are of great importance: the type of anesthesia and the simplicity of the method adopted for emptying the uterus. It is essential to avoid the likelihood of sepsis, and yet to place as slight a tax as possible upon the already burdened organism. At any time during the first sixteen weeks of pregnancy, dilatation of the cervix and curettage of the uterus under spinal anesthesia has proved a simple and rapid method of performing abortion.

From the fourth to the seventh month of gestation, termination of pregnancy may be readily brought about by packing the cervical canal with gauze, after sufficient mechanical dilatation. Within twenty-four hours the fetus and placenta are expelled or can be easily removed. The likelihood of sepsis or hemorrhage is small.

When the complication of carcinoma of the uterus or the presence of a large fibroid is added to that of tuberculosis during pregnancy, hysterectomy will be required to care for these contingencies, and will at the same time encompass the necessary abortion. Unless some such condition arises in the pregnant woman requiring the removal of the uterus, this procedure should be avoided as too drastic.

The effect of abortion in a case of pulmonary tuberculosis depends largely upon the method adopted and the time in the pregnancy at which it is undertaken. In some cases the specific reaction of the patient to the operation is slight, but at other times in spite of the rapidity and skill with which it is accomplished, there may be very definite repercussions which endure for weeks, such as an unstable nervous system, accelerated heart action, secondary anemia, and a general lowering of the patient's vitality, with a resulting increase in the extent of the pulmonary lesion. In the great majority of cases, however, where abortion is relied upon to contribute in checking the progress of a tuberculous process which cannot be controlled by pneumothorax, the patient is spared the tax upon her circulation, the rapid descent of the diaphragm following labor, the exhaustion and loss of blood accompanying parturition, as well as the responsibilities of caring for the infant.

STERILIZATION

In view of the increased sexual activity common among the tuberculous, and the resulting likelihood of pregnancies, the question of sterilization frequently arises. The physician has only partly performed his duty to his patient by interrupting a single pregnancy, as the possibility of invalidism or even death from tuberculosis may be a recurrent issue with each succeeding confinement. Sterilization is the only trustworthy method of controlling this situation. If this course is determined upon, the time at which it should be undertaken will be decided upon both medical and practical considerations. Ordinarily a tuberculous patient is in such a condition when abortion is resorted to that her state of health makes it inadvisable to proceed to sterilization at one and the same time. From the strictly medical point of view, therefore, it is usually better to wait until the patient has recovered from the effect of the emptying of the uterus, and the tuberculous process has been brought under control. As a practical matter, however, in view of the

likelihood of renewed conceptions, unless the patient be under surveillance in an institution, it is frequently desirable to proceed at once to sterilization.

With the progress that is being made in sterilization by x-ray, it is to be hoped that this will present a dependable method within the near future, as it seems to have none of the ill effects inherent in surgical procedures. At the present time, however, surgery affords the most reliable means. The possible operations are oöphorectomy, salpingectomy, hysterectomy and a combination of two or more. The method chosen should be the one that places the least tax upon the woman, bearing in mind that the selection of an anesthetic is important in every case of pulmonary tuberculosis. Ordinarily, the most satisfactory is that which consists in the removal of a small portion of each fallopian tube and the suturing of the cut ends. This operation may be successfully accomplished under either spinal or rectal anesthesia.

A new possibility is presented by recent experiments with the immunization of women by the hypodermic injection of semen as an antigen. This appears to have the great advantage of affording temporary loss of fertility, which can be controlled at will.

The field of prophylactic sterilization is not narrowed to include only the tuberculous woman. The man, not burdened with phthisis, nor yet a subject for major surgical procedure because of the accessibility of the organs of generation, can be easily rendered nonproductive.

The operation in common use is that of isolating the vasa deferentia and excising a small portion of each. The cut ends are then sutured. There is no danger of shock or of hemorrhage and the man's sexual activities are not impaired.

Radiation by x-ray will produce aspermatism. How long this continues is a debatable question, but as the result of thorough exposure it has been found to persist for months or even years.

CONCLUSIONS

The treatment of pulmonary tuberculosis during pregnancy demands active measures directed to control the tuberculous process, and the use of some form of therapeutic collapse in suitable cases is often successful in bringing about an arrest of the phthisical lesion. The tax upon the patient's vitality, kidney function, and lung capacity, brought about by the complication of pregnancy, has a direct bearing upon the course of pulmonary tuberculosis. Unless the pulmonary lesion is relieved by pneumothorax or phrenicotomy, the cessation of pregnancy may be brought about by the surgical removal of the fetus.

Therapeutic abortion is not so widely employed as in former years, nevertheless, where it is resorted to, the favorable or unfavorable effect upon the patient stands in direct relation to the stage of gestation in which it is performed, the choice of the anesthetic, and the simplicity of the surgical procedure.

Sterilization, at the time of the abortion, or at a later period, may be necessary to prevent pulmonary disintegration or even death from phthisis contingent upon recurrent pregnancies.

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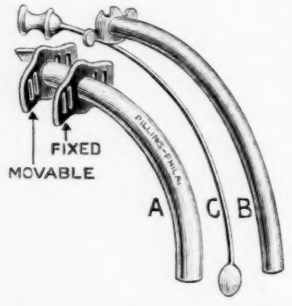
A MODIFIED TRACHEOTOMY TUBE

BY LE ROY A. SCHALL, M.D.*

FOLLOWING tracheotomy, there is a variable period during which the tracheal secretion is



very profuse. During this time, the dressing becomes soaked with secretion and requires frequent changing, necessitating almost constant nursing care. In changing the dressing, the tracheotomy tube is frequently moved, which causes spasms of cough with consequent expulsion of more mucus.

To overcome this difficulty, this tracheotomy tube was devised. It is essentially an extended Jackson tube with a double neck plate; the inner fixed, the outer movable. The tube is inserted in the usual manner, but the extension permits more gauze to be applied without disturbing the curve or fit of the tube.

By its use the tracheal wound is kept drier, therefore cleaner; the patient is more comfortable as he is disturbed less; and less nursing care is required.

When the tracheal secretions cease, the tube may be replaced by any of the other types of tubes which the case may indicate.

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HEALTH OFFICERS' MONTHLY STATEMENT OF VENEREAL DISEASES REPORTED

TREASURY DEPARTMENT—PUBLIC HEALTH SERVICE

DECEMBER, 1934

This statement is issued monthly for the information of health officers in order to furnish current data as to the prevalence of the venereal diseases. The following reports were received from State Health Officers. The figures are preliminary and subject to correction. It is hoped that this will stimulate more complete reporting of these diseases.

State	Syphilis: Cases Reported During Month	Syphilis: Monthly Case Rates per 10,000 Population	Gonorrhea: Cases Reported During Month	Gonorrhea: Monthly Case Rates per 10,000 Population
Alabama (a)	—	—	—	—
Arizona	22	0.49	155	3.42
Arkansas (c)	298	1.59	215	1.15
California	1,160	1.91	1,245	2.05
Colorado (a)	—	—	—	—
Connecticut	50	0.30	61	0.37
Delaware	140	5.81	31	1.29
Dist. of Columbia	127	2.57	88	1.78
Florida	349	2.25	55	0.35
Georgia	583	2.00	350	1.20
Idaho	0	—	0	—
Illinois	1,242	1.59	1,053	1.35
Indiana	178	0.54	72	0.22
Iowa (c)	151	0.61	125	0.50
Kansas	100	0.53	80	0.42
Kentucky	177	0.67	218	0.83
Louisiana	156	0.72	82	0.38
Maine	40	0.50	34	0.42
Maryland	669	4.02	190	1.14
Massachusetts	383	0.89	570	1.32
Michigan	546	1.08	547	1.08
Minnesota	322	1.24	289	1.11
Mississippi	932	4.55	1,549	7.57
Missouri	645	1.76	363	0.99
Montana (c)	35	0.65	33	0.61
Nebraska	27	0.19	51	0.37
Nevada (a)	—	—	—	—
New Hampshire	13	0.28	12	0.26
New Jersey	492	1.17	250	0.60
New Mexico (c)	51	1.18	41	0.94
New York	4,507	3.48	1,157	0.89
North Carolina	830	2.53	235	0.72
North Dakota	22	0.32	38	0.55
Ohio (c)	685	1.01	340	0.50
Oklahoma (c)	246	1.18	155	0.74
Oregon	34	0.35	81	0.82
Pennsylvania	261	0.27	199	0.20
Rhode Island	127	1.81	102	1.45
South Carolina (c)	220	1.26	353	2.02
South Dakota	5	0.07	28	0.40
Tennessee	1,090	4.09	582	2.18
Texas	447	0.74	161	0.27
Utah (a)	—	—	—	—
Vermont	23	0.64	20	0.55
Virginia	389	1.59	243	1.00
Washington	207	1.29	197	1.23
West Virginia (b)				
Wisconsin (d)	114	0.38	21	0.07
Wyoming (a)	—	—	—	—
Total	18,095	1.52	11,671	0.98

(a) Not reporting.

(b) Has been reporting regularly but no report received for current month.

(c) Incomplete.

(d) Only cases of syphilis in the infectious stage are reported.

Survey in which all medical sources have been contacted in representative communities throughout the United States have revealed that the monthly rate per 10,000 population is 6.6 for syphilis and 10.2 for gonorrhea.

VERMONT STATE MEDICAL SOCIETY

THE NEPHRITIDES*

BY ALBERT A. EPSTEIN, M.D.†

THE subject which you have invited me to consider is a very large and perplexing one. As evidence I offer the simple fact that my friend and colleague Dr. Fishberg, who has already addressed you, has spent several years and some six hundred pages thrice edited, in an attempt to elucidate it. Therefore, to touch upon even a few of the high spots of the subject in a brief symposium, is a very ambitious undertaking. As I have no intention of detaining you any longer than the time allotted me, I trust that you will be indulgent if I omit some points in my discussion.

Our knowledge of nephritis has passed through several evolutionary stages: the clinical, the pathological, the experimental, and the functional. As a result much information has been amassed and a vast literature exists on every phase of the subject. When the presence of albumin and of casts were regarded as the basic signs of renal disease, the concept of nephritis was much simpler than it is to-day.

The efforts of pathologists and clinicians to coördinate the morbid processes of the kidney with its clinical manifestations, have created a good deal of confusion which has not been entirely dispelled even by the more recent advances in our knowledge.

A proper understanding of nephritis now requires a foreknowledge of pathology, physiology, chemistry and general clinical medicine. Without these fundamentals an adequate comprehension of nephritis is extremely difficult because of the problem of correlating the information which we gather by the different methods of analysis and investigation.

Pathology has taught us the various forms which disease of the kidneys may assume and has established their relationship to diseased processes in other parts of the body. But, the pathological concept alone has proved inadequate owing to its frequent failure to correspond with clinical manifestations and other findings. By virtue of this fact we now recognize that kidney changes may be the effect as well as the cause of many of the phenomena commonly associated with renal disease. By way of example I wish to call to your attention the reciprocal relation which exists between the cardiovascular system and the kidneys, for it is now conceded that, while cardiovascular damage may ensue from, or be concomitant with renal disease, the converse is also true; namely, pathologic changes in the blood vessels may eventuate in disease of the kidneys. Realization of this is important, not only in connection with proper diagnosis, but in the application of therapeutic measures.

From experimental procedures on animals we have learned much concerning the intimate relationship between certain pathologic changes and the physiologic alterations of the kidneys. The unqualified application of this knowledge to the clinical interpretation of renal disease is not always admissible for the reason that the conditions under which nephritis is produced experimentally, and those under which it arises clinically are not comparable. I have in mind particularly the work of those investigators who attempted to stress the functional element in the clinical concept of nephritis, and were responsible for a host of tests, many of which have now been completely discarded. I do not mean to imply that functional considerations are no longer important. Quite the contrary, I merely wish to indicate that the application of functional data in the interpretation of renal disorders must be extremely guarded because the position of the kidneys in the animal economy is such that alterations in renal function may arise from causes outside the kidney. This is particularly well illustrated in the hydropic states which are frequently associated with disease of the kidneys, and which are directly traceable to extrarenal factors. Failure to realize this fact has retarded our understanding of many conditions associated with renal disturbances, for more than three-quarters of a century, and prevented the application of suitable therapeutic measures in the management of such cases.

It has become quite clear that the clinical syndromes of the various types of nephritis cannot be regarded in the light of renal pathology alone. Pathologically speaking, nephritis is an inflammatory process of the kidneys. In reality however, the clinical concept of nephritis includes not only a variety of diseased processes, some of which are not of an inflammatory nature, but it embraces also many abnormal conditions affecting nearly every organ and system of the body.

On the basis of our present knowledge then, the comprehension of any given case of nephritis is dependent upon three distinct factors: first, the determination of the pathologic processes involved; secondly, the evaluation of kidney function, and, lastly, the appraisal of

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extrarenal manifestations which contribute to the sum total of the clinical phenomena presented. An accurate diagnosis of nephritis therefore demands a circumspect and complete analysis of all the morbid conditions present, the etiologic factors involved, the disturbance in function and other disorders which arise therefrom.

Although the anatomical classification of kidney diseases is not wholly adequate in all cases, still we must nevertheless rely upon it for guidance, because etiologic and functional classifications aid us but little and are not practical.

The types of nephritis which first require our attention are the "acute forms", namely, the acute glomerulonephritis, the acute nonsuppurative interstitial nephritis, and the acute tubular nephritis or "nephrosis". Any one of these may constitute the forerunner of the contracted kidney or the large white kidney.

The problem in the diagnosis of acute nephritis is essentially different from that of chronic nephritis. In acute nephritis we have to deal with acute damage to normally functioning organs, which previously sound, are again more or less quickly restored to normal, provided they are not overwhelmed with the destructive agent. In the diagnosis of acute nephritis or of subacute nephritis, a consideration of the etiology is very important. This can usually be traced to bacteria or their toxins, or, as I believe to be the case in certain types, to constitutional disorders of metabolic or endocrine origin.

The intimate connection between infective conditions and nephritis is very well known. In acute nephritis due to the action of bacterial toxins, the renal reaction is a diffuse inflammatory process. The onset is rapid and the signs are unmistakable. Occasionally nephritis may result from actual lodgment of bacteria in the kidney substance (bacterial thrombi), giving rise, in consequence, to a disseminated focal type of lesion of which a good example is the embolic glomerulonephritis of subacute bacterial endocarditis. The diagnosis frequently depends upon the recognition of the original disease. In the more fulminating types of general infections, such as those of postpartum sepsis, or suppurative sinus thrombosis, cocci may appear in the urine associated with blood elements, pus, and granular casts. The diagnosis in such cases is self-evident. I need not discuss the clinical features of each of the acute forms. They are well known.

The existence of acute nephritis, excepting of course the chemical nephritides and the types occurring in pregnancy, therefore points usually to an antecedent infection. But renal disorders with urinary signs frequently occur in febrile diseases of all kinds which do not represent full-fledged nephritis, and thus the problem of differentiation often arises. In this latter group of cases, of course, the signs usually are not so pronounced. There is an albuminuria, at times with casts. Functional disorders, as we shall see later, also arise. But there is not, as a rule, that marked evidence of renal involvement which we find in the true cases of nephritis; nor does the disturbance last much beyond the duration of the febrile state. The difference, perhaps, is arbitrary, and one of degree only.

However, the tendency of uncomplicated acute nephritis is also to recovery, provided, of course, infective factors be no longer present and active to work new damage to the kidney. This is well shown in clinical experience by the complete disappearance of the nephritis of scarlet fever cases.

In persistent or recurring infections the condition may go on to chronicity. Present day experience with infected tonsils and sinuses as well as infections about the teeth may account for many such instances. Many cases of chronic nephritis apparently following the acute lesion have been reported, and an antecedent scarlet fever may be an important historical fact in the diagnosis. In my own experience, however, this causal factor seems to play less of a part than hitherto ascribed to it.

In the matter of chronic nephritis, the problem of etiology as a source of information is much more difficult. No doubt in a certain number of cases a history of acute nephritis or recurring infections may be elicited and a diagnosis arrived at, but the connection between the two is not always clear. At this point, I wish to call particular attention to a group of cases, of which I have seen many examples, and of which no mention appears in the literature. It concerns individuals with a seborrheic type of skin, who suffer from frequent or intractable acne of the face and body. They are particularly prone to recurrent attacks of acute nephritis which ultimately terminate in a rapidly progressive or malignant form of chronic nephritis. The clinical course of the malady in such individuals seems to be so constant that I would venture to say, that whenever one is seen in the first attack of acute nephritis, it is safe to predict that other attacks will follow, even though the first one may subside entirely. It is therefore imperative to remember that infections of the skin, even such a one as acne, may play a most important part in the causation of renal disease.

Furthermore, chronic poisoning of one sort or another, tuberculosis or syphilis may be contributory factors and require special consideration in establishing a diagnosis.

There is one difficulty that presents itself from the clinical side in investigating the question of the connection between acute, subacute and chronic nephritis, and that is, as already intimated, that acute and subacute nephritis

may appear during the course of an infection without the development of symptoms other than the urinary signs. Moreover, in the chronic nephropathies, it is the possibility and frequent occurrence of compensatory processes which create some difficulty in establishing a diagnosis. This is particularly true when the question is viewed from the functional standpoint. It is noteworthy in this connection, that careful and frequent observation of the concentrating power of the kidneys together with a measure of the urinary output, is often of great assistance in arriving at a conclusion.

Notwithstanding the efforts of pathologists and others, the diagnosis of chronic nephritis still is, as it has been in the past, largely conventional. While a correct anatomic diagnosis is always desirable, from the standpoint of the practitioner, it is not always of especial importance, except in such cases where a differentiation is necessary of factors due to renal disease, and those due to disease in other organs. I must take issue with those whose main clinical object in the study of nephritis appears to be its classification. To the practitioner this insistence has been and still is a source of perplexity and great confusion. Our aim in the diagnosis of the nephritides should not be merely to establish the pathologic character of the disease, but to ascertain the facts which are vital in its treatment.

Chronic nephritis presents a great variety of symptom combinations. It would be impossible to consider them all in this brief discussion. However, certain clinical types occur with such regularity, that definite rules may be established for their recognition. Thus, the chronic nephritides may be divided into two general classes: (1) the azotemic, to which belong the chronic interstitial forms; primary and secondary, in which retention of nitrogenous waste products in the blood occurs leading to and terminating in uremia; (2) the hydropic, to which belong the chronic forms and the nephroses, and which are characterized by the retention of water and salt, and the development of edema. Nevertheless in certain cases the clinician finds himself embarrassed when it comes to appraising the relative value of the heart, the blood vessels, and the kidneys, in the production of some of the symptoms.

Albuminuria, with or without casts, is still the cardinal point about which all questions of renal diagnosis revolve. The glomeruli constitute the filtering apparatus of the kidney. Under ordinary circumstances water, salt and other crystalloids are permitted to go through, while the proteins of the blood (or colloids) are prevented from going through. Damage to the kidneys manifests itself not only in the rate of excretion or filtration of the water and soluble material, but also by changing its attitude toward the colloids, namely, the albumins or proteins of the blood. This is the origin of albuminuria. It is generally stated that the excretion of albumin is the result of an increased permeability of the glomerular capillaries. While this may be so, I am of the opinion, that under certain circumstances, changes in the permeability of the renal capillaries need not be the primary cause of an albuminuria. I believe that in certain instances an alteration in the biochemical character of the blood proteins may occur, so that they behave as foreign material and are permitted to go through. This mechanism is operative, I believe, in causing the albuminuria in certain renal disorders (the nephroses). That an increased permeability of the glomerular capillaries is necessary to accomplish this, is only of secondary importance. As yet, pathologic investigation is not sufficiently refined to prove conclusively just what is meant by increased permeability.

Given a case with albumin in the urine, the answer must be given whether it portends a nephritis. Is it the expression of a definite renal lesion, or some other disorder? Inquiry must be made into the history for evidences of infection, or intoxication, by drugs and other agents, syphilis, recent pregnancy, etc., to establish, if possible, a traceable etiology. The discovery of an etiologic factor does not necessarily determine the diagnosis of nephritis, but it furnishes good presumptive evidence.

In children and young adults the problem frequently arises of the differentiation of postural albuminuria. This is a common contingency, and the presence of a marked albuminuria, without casts, or with rare casts, in a young person should always suggest the possibility of its being of this peculiar type. This point cannot be stressed too much. We know that examination of the separate urines passed at different times of the day and testing the effect of standing fifteen to thirty minutes in the lordotic position, will often clear up the diagnosis promptly. The large amount of protein precipitable by acetic acid in the cold is an important feature of the albuminous urine in these cases. A similar chemical reaction, however, is frequently obtained in the albuminuria which occurs in association with glycosuria in diabetes. Other evidences of renal disease in these cases may be entirely wanting, but the albuminuria does not seem to be of postural origin.

Aside from these and the other group, there is a large number of patients in whom no assignable cause for the albuminuria can be found. Local causes in the urinary tract may be responsible for the albuminuria (stones, tumors and other conditions). These conditions, of course, should be excluded.

Although our knowledge in this matter is not complete, we know that certain anatomic peculiarities of diseased kidneys are character-

istic of different operating causes. So that etiology may not only aid us in determining the presence of renal disease, when the cause of the albuminuria is established, but also in divining the exact nature of the trouble.

If we wish to gather all possible clinical data we will not rest content with historical facts, nor with an anatomic diagnosis of nephritis, but will proceed to determine the degree to which the kidney function has suffered.

Generally speaking, the functional methods of renal diagnosis are divisible into two groups: (1) those which aim to ascertain the character and extent of renal disease by estimating the response of the kidneys to physiologic stimuli; (2) the other aims to determine the nature and degree of the damage, arising from the insufficiency of the kidneys.

To this first group belong those tests which depend on the excretion of substances administered in known quantity, which are recoverable more or less completely from the urine. To the second belong the direct observations on the blood and urine.

Many considerations render the study of renal function a very complex problem. It is a mistake to regard any one functional test as being capable of measuring renal function as a whole, as each test at best covers only a limited range of kidney activity. To maintain normal conditions in the body the kidneys need perform but one function, namely, adequate filtration. This they can achieve by excreting a limited, but suitable amount of concentrated urine; or, by excreting a large amount of dilute urine. The factor of safety in this respect is indeed very great. As stated before, the glomerulus is the filtering apparatus of the kidney. The number of the glomeruli in the two kidneys is very great, and we know that only a limited number of them are at work at any one time. It has been definitely established by experiment, that the glomerular content of about one-third of one kidney is sufficient to maintain the excretory needs of the body in normal equilibrium.

Experience has shown that the most constant effect upon kidney function, arising from chronic disease within it, is its inability to concentrate urine. This disability, as has already been intimated, may be largely overcome by a compensatory increase in the amount of urine excreted, and no ill effect need follow. It is only when both the concentrating power and the compensatory diuresis fail, that dire results ensue.

The normal function of the kidneys comprises among other things, the elimination of the waste products of nitrogenous metabolism. Failure on the part of the kidneys to function properly causes ultimately a retention of these substances in the body and their accumulation in the blood. The tests measure one or another of these substances; all of which come under the heading of incoagulable or nonprotein nitrogen. The level of the nonprotein nitrogen of the blood must be determined as the resultant of at least three factors: namely, kidney efficiency, diet, and protein destruction. In appraising kidney function on this basis, due credit must be given to each of these factors. One other factor, usually ignored, is of the greatest importance, and that is the fact that, in renal conditions associated with edema, much of the retained nitrogenous waste products are distributed throughout the body in the retained fluid, so that the blood does not show the same concentration that it would, had there been no edema. It is not uncommon to find in such cases, that the nonprotein nitrogen in the blood increases as the edema subsides.

Certain investigators lay the greatest stress on accumulation of urea in the blood; others believe that uric acid and creatinin give the earliest and best indication of renal incompetency and disease. For my part, I believe that the nonprotein nitrogen as a whole or its one component urea, gives the best indication of renal disease. In the absence of edema, a persistently low nonprotein nitrogen or urea in the blood, does not signify that nephritis is not present, but it does indicate that the functional capacity of the kidneys is adequate for the excretion of the waste products from the blood.

As a prerequisite to the proper interpretation of the results obtained in the study of renal function, extrarenal factors which are capable of modifying or influencing them, should be definitely excluded. The reasons for this are manifold.

It is generally known that cardiac insufficiency may give urinary signs like those of renal disease, and functional tests may also yield evidence to that effect. Following febrile conditions, renal function may remain disturbed for some time after the fever has subsided. In severe or pernicious anemia, the kidneys may behave in very much the same way as they do in nephritis.

There is one group of cases in which the renal function is extremely deranged, particularly as it relates to water and salt excretion, and yet the cause of the disturbance resides largely, if not wholly, outside the kidney. I refer here to the type known as "chronic nephrosis". It is characterized clinically by an oliguria and intense albuminuria, with a tendency to the development of edema.

I have just spoken of oliguria and edema and intimated that they may be of extrarenal origin. As these two phenomena occur frequently in association with renal affections, further elucidation may be in order. Oliguria, or diminished urinary excretion, may result from a variety of

causes which directly or indirectly affect the kidney, as for example: inflammation, as in acute nephritis, congestion as in circulatory disturbances arising from interference with venous blood flow, due either to pressure, portal thrombosis, cirrhosis of the liver, circulatory collapse or limited availability of water for excretion due to alterations in the chemical composition of the blood. This last element is of importance in all instances where the protein or colloid content of the blood is diminished with consequent reduction of the osmotic pressure of the blood; a condition which permits the flow of water into the tissues, and thereby prevents the kidney from excreting it. Hand in hand with the factors concerned in the production of oliguria, are those responsible for edema. When the kidneys become affected, as by acute inflammation or congestion, and the urinary excretion becomes reduced, fluid is gradually retained in the body. At first it may be imperceptible, but gradually, as the condition progresses, deposits of fluid appear under the skin or in the serous cavities. Some of this may be due to actual inability of the kidneys to eliminate fluid; some, however, may result from damage or engorgement of the blood capillaries. By virtue of the increased pressure within them, some of the fluid content of the blood filters through into the tissue spaces. As evidence of this, is the fact that the edema which develops under such circumstances is composed of a fluid which resembles the serum of the blood in many respects.

However, fluid under the skin and in the serous cavities may be deposited by a different mechanism; one in which neither the kidneys nor the circulatory apparatus are directly concerned.

The proteins of the blood serum are greatly concerned with the regulation of fluid exchange between the blood and the tissues. Normally the blood serum contains six to eight grams of protein per 100 cc.; at least five grams per 100 cc. are necessary to maintain the fluid exchange in equilibrium. When the protein content falls below this level, the blood loses its optimum osmotic pressure, the fluid exchange becomes unbalanced, and the flow of water and its dissolved salts is from the blood stream in the direction of the tissues. In consequence of this, subcutaneous edema and effusion into the serous cavities take place. The kidneys actually have nothing to do with this process. Because of the mechanism involved (reduced osmotic pressure) the edema and serous effusions are almost water clear, and contain little or only traces of protein.

It is this type of edema that occurs characteristically in the condition known as "nephrosis". The underlying cause is the reduced protein content of the blood serum, conditioned by an excessive loss through the kidneys, i.e., the albuminuria. Loss of protein from the blood from any cause or in any manner which impoverishes it to a sufficient degree is capable of inducing this type of edema. It is by virtue of this fact that a variety of nephritic conditions with intense and prolonged albuminurias, develop in due course an edema of this character, and are designated as cases of "nephritis with the nephrotic component".

Of course profound loss of protein from the blood is accompanied by other changes in the composition of the blood, for instance, change in the relation of the individual fractions of the serum protein, and in the content of fatty or lipoid material which generally becomes greatly increased. It is my belief that a real difference exists between the true cases of "nephrosis" and those cases of "nephritis with the nephrotic component", namely, that in the true "nephrosis" the blood changes mentioned appear earlier, and that they are more profound. Certain clinical differences exist between these two conditions: (1) the urinary findings characteristic of renal inflammation are usually lacking in "true nephrosis", and (2) changes in the cardiovascular system, in the eye grounds, etc., are also wanting. So that under ordinary circumstances the distinction can be made readily. Time will not permit me to go into further details; I might state in passing, however, that differences in age incidence and the absence of any determinable etiology are often helpful in establishing a diagnosis of "nephrosis".

Apart from the academic interest of any disease, the question which interests the practitioner most is treatment, and I cannot conclude my discourse without a few words on this subject. It is very gratifying to note the realization by most men in our profession, that nephritis is not a disease entirely restricted to the kidneys, and no specific therapeutic measures which can be directed to the kidneys are available.

In the matter of the acute nephritides and those forms of chronic nephritis which result from recurrent attacks of the acute varieties, preventive measures, i.e., the elimination of the causes which operate in their production, should be applied. Foremost among these causes are the various intoxications (and in these may be included pregnancy with eclamptic tendency), and the different general and focal infections. Inasmuch as the acute nephritides are usually self-limited, and for the most part run a favorable course, the palliative measures are few and simple. General hygienic procedures, as well as sparing the kidneys any unnecessary work from improper nutrition, such as limitation of protein and salt intake, are almost always sufficient. Mild alkalinization may be in order, but symptomatic treatment is rarely acquired, and the use of drugs to promote the

elimination of urine should be entirely interdicted. A general "hands off" policy is, more often than not, the best course to pursue.

Of course there are many instances in which an acute nephritis does not subside completely; certain others which do not progress into slow and gradual chronicity are rather fulminating in character with marked cardiovascular changes and renal insufficiency, leading to profound intoxication and ultimate uremia. The existence of edema, contrary to general opinion, is not an untoward symptom, and in the presence of a severe azotemia should not be combated. The therapy of such cases is extremely difficult. Measures which promote vasodilation, counterirritation, phlebotomies, infusions of glucose, and on occasions decapsulation may be of benefit. This latter procedure does not, however, often prove beneficial and should be resorted to only in extreme situations. Blood transfusions are not desirable, in fact in my opinion are definitely contraindicated.

With the exception of the nephroses, the chronic nephritides present somewhat different problems, although in the main, the principles involved in their treatment are the same as those underlying the treatment of the fulminating varieties of the acute forms. They are symptomatic and palliative in character. The subject is rather simple when considered in three stages: the first relates to the defective kidney function and its effects upon the composition of the blood; the second is concerned with the cardiovascular changes and their sequelae; and, the third involves the retention of water, i.e., edema.

The methods which are most serviceable in combating these three sets of symptom complexes of chronic nephritis are undoubtedly known to you all, and it would be prolix to attempt to describe them. Above all a common sense attitude is necessary, and it is well to remember that the chief functional requisites of the kidneys are the elimination of water and salt and the nitrogenous waste products. Inasmuch as the clinical picture which any case presents is usually the composite of symptoms arising both from renal and extrarenal causes, the therapeutic measures employed must be directed toward the different components of the clinical complex. If it is the azotemia which is paramount, then its source, namely, protein intake, must be reduced to a minimum. Nitrogenous waste from tissue deterioration is best controlled by liberal carbohydrate administration in any and every form. Abundant fluid administration may also be useful when water excretion by the kidneys is unaffected.

The symptoms which arise from cardiovascular changes (whether primary or secondary) are best controlled by those agents which reduce and maintain the blood pressure at a safe level, and preserve the competency of the heart.

Water retention, as stated above, may be of renal, circulatory, or blood origin. The methods employed to overcome it must be suited to the mechanism which causes the retention. It should be remembered that medicinal agents calculated to aid the function of the kidneys are usually ineffectual, and should be avoided. So that, when water retention is of renal origin, vasodilators, phlebotomy and counterirritation should be used, but only in such instances where there is no co-existing azotemia. When azotemia is present, the retention of water in the body is often a useful circumstance and delays the development of uremia. Water retention due to circulatory failure should be treated with measures appropriate to such a condition, but the use of mercurials should be avoided.

By retention of water of blood origin, I mean the hydropic state which results from the reduced protein content of the blood serum conditioned by an intense and protracted albuminuria. This, as already stated, is encountered in nephrosis, and in nephritis with the nephrotic component. The medication in both sets of conditions is, of course, the restoration of the protein content of the blood serum to normal. This is best achieved by the administration of large amounts of protein in the diet. This principle is applicable in all cases where the protein content of the blood stream is reduced, with the exception of those in which a marked azotemia is also present. In such cases the protein intake in the diet should be maintained at the basal level, particularly if the specific gravity of the urine is persistently and consistently low. It is better under such conditions to have an edematous than a uremic patient.

In nephrosis the administration of certain agents which promote water excretion such as the mercurials, urea, ammonium chloride and calcium salts, is permissible. But, as I have reiterated on repeated occasions, the object in the treatment of the nephroses should be not only the promotion of water excretion and the removal of edema, but an earnest effort should be made to cure the condition. In certain cases the administration of large amounts of protein together with the use of adequate doses of thyroid, must be persisted in over long periods of time in order to attain a result.

The expression "last but not least" does not apply to the question of prognosis. For my own part the less said about it the better. The cases of acute nephritis are usually self-limited and offer a favorable outlook, particularly when the cause is established and removed. With the chronic nephritides the case is different. Our therapeutic attainments leave much to be desired, and I do not know of any method or device by which an exact prognosis can be formulated. It is only in the case of true neph-

rosis that the outlook is good. Barring complications (such as erysipelas and peritonitis of pneumococcic origin) which are not infrequent, it is my firm conviction that the condition is not only amenable to satisfactory treatment but is capable of complete cure.

MISCELLANY

VERMONT DEPARTMENT OF PUBLIC HEALTH

JANUARY, 1935

The incidence of communicable diseases during January is as follows: chicken pox 216, diphtheria 3, infantile paralysis 2, measles 91, mumps 6, scarlet fever 114, undulant fever 1, tuberculosis 5 and whooping cough 302.

The Laboratory of Hygiene made 1,663 examinations during the month, classified in the following manner:

Examinations for diphtheria bacilli	224
" " Widal reaction of typhoid fever	34
" " undulant fever	40
" " gonococci in pus	139
" " tubercle bacilli	184
" " syphilis	554
" of water, bacteriological	181
Examinations of water, chemical and bacteriological	29
" " milk, market	191
" " milk, submitted for chemical only	11
" " milk, submitted for microscopical only	0
" " foods	15
" " drugs	0
" for courts, autopsies	2
" " courts, miscellaneous	6
" miscellaneous	53
Autopsies to complete death returns	0

Twenty-nine cases of gonorrhea and nineteen cases of syphilis were reported to the Division of Venereal Diseases during January. One thousand one hundred and twenty-four Wassermann outfits and 402 slides were distributed by this Division.

The nurses of the Poliomyelitis After-Care Division made forty-three home calls, four social calls, visited six doctors and made fifty-three visits during the month. Five pieces of apparatus were fitted to patients, four orthopedic corrections made to shoes, two patients admitted and one patient discharged from the Children's Hospital. Eight patients were admitted and two discharged from the Audubon Hospital and one patient discharged from the Massachusetts General Hospital.

The vocational worker of this Division reports sales made to the amount of $109.08 for the month.

YALE MEDICAL SCHOOL GRADUATE WINS PEDIATRIC PRIZE

Dr. Leona Baumgartner, who graduated last June from the Yale University School of Medicine, has been awarded the prize offered by the New England Pediatric Society for the best paper presented last year by fourth-year medical students in New England on a subject of scientific interest in connection with the health of children. The subject of her paper was "Age and Antibody Production."

The award to Dr. Baumgartner was the fifth successively to be won by Yale medical students. Dr. Jacob Greenberg won the prize in 1933, Dr. David J. Cohen in 1932, Dr. Abraham J. Schechter in 1931 and Dr. Robert D. M. Cunningham in 1930. The amount of the prize is $100.

Dr. Baumgartner graduated from the University of Kansas in 1923, obtained a Ph.D. degree at Yale in 1932, and an M.D. degree in 1934. She is now an intern at the New York Hospital.

THE APPOINTMENT OF DR. ETHEL C. DUNHAM

Dr. Ethel C. Dunham, a member of the staff of the Department of Pediatrics of the Yale University School of Medicine since 1919, has been made acting director of the Division of Maternal and Child Health, United States Children's Bureau. In her new position Dr. Dunham will be associated with Dr. Martha M. Eliot, a former member of the Yale Pediatric Staff, who was recently appointed assistant chief of the United States Children's Bureau.

Dr. Dunham is widely known as an investigator in the field of child health. For the past eight years she has devoted much of her time to research on the diseases of newborn infants. Her work has been effective in directing attention to the causes of infant deaths in the prenatal period and to preventive measures.

Dr. Dunham was one of the first women to be elected to membership in the American Pediatric Society, and is chairman of the Society's committee on the study of the prematurely born child. She also holds membership in the American Academy of Pediatrics. She obtained her medical degree at Johns Hopkins. After spending one year at the Johns Hopkins Hospital as resident house officer, she came to the New Haven Hospital where she has since risen to the rank of associate clinical professor of Pediatrics in the University and attending pediatrician to the New Haven Hospital.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.

CASE 21091

PRESENTATION OF CASE

Approximately five months before entry the patient, a thirty-three year old single stenographer, experienced an attack of sharp midabdominal pain which lasted for several hours. This was not accompanied by nausea, vomiting or other gastro-intestinal disturbances. She remained in bed for two days. On the morning of admission while still in bed she suddenly developed severe, non-radiating, midabdominal pain. She felt hot but did not vomit. Her physician who was called late that day advised immediate hospitalization.

Her family history is non-contributory.

Fifteen years before entry she had a severe attack of rheumatic fever following which she developed a slight cardiac lesion. She had a tonsillectomy nineteen years before entry. Her menstrual periods were always regular, lasting five or six days, and always associated with severe cramp-like pains. There was a slight discharge preceding each period. At the time of admission she was in the midst of a menstrual period and had lower abdominal cramp-like pains.

Physical examination showed a well-developed and nourished woman lying quietly in bed. The chest was negative. There was a late, rough, diastolic murmur at the apex. The lower midabdomen showed moderate tenderness. The abdomen was soft and level. No organs were felt.

The temperature was 101°, the pulse 88. The respirations were 22.

Examination of the urine was negative. Examination of the blood showed a white cell count of 17,400.

At operation, on the day of admission, the appendix was found in the pelvis. Scattered over the peritoneal surfaces of the large and small bowel, as well as on the parietal peritoneum, were flecks of brownish mucoid material. This was especially marked in the region of the appendix. This material could be rubbed off the bowel in places, but in others it seemed to be in a thick layer and did not yield to rubbing. The appendix did not appear reddened, but its distal half was firm and nodular. It was removed. Further exploration of the pelvis showed a normal uterus. The right ovary was slightly enlarged but not otherwise abnormal. The left ovary was not very well seen but as nearly as could be determined was normal to palpation. There was a slight amount of turbid fluid in the pelvis. She did well postoperatively and was discharged on the thirteenth day.

DIFFERENTIAL DIAGNOSIS

DR. JOE V. MEIGS: "She remained in bed for two days." This statement makes me think that this patient's discomfort must have been a real attack of something. After a second attack her physician advised immediate hospitalization. Again she must have had a real attack of something for her physician apparently thought that she should be under cover in a hospital so she could be operated on if necessary. This sharp, sudden pain may possibly have been appendiceal in origin, but one should think of a rupture of some viscus which came on suddenly; perhaps something broke which caused acute pain, or else she had a stone of some kind.

Her cardiac lesion makes one think of a possible embolus in the mesentery which might have caused the attacks of pain that she had had.

Her menstrual history is quite negative. She had a moderate amount of discharge before the onset of the menstrual period. That is not unusual, rather more usual than not.

"At the time of admission she was in the midst of a menstrual period and had lower abdominal cramp-like pain." I do not see how we can use that information at all. She had pain and also had menstrual pain, and I think it would be very difficult to differentiate that and another pain. However, if this pain were different from the menstrual pain it might possibly be due to bleeding from a ruptured ovary such as a ruptured follicle or ruptured corpus luteum. It is unusual to find this happening at the time of the menstrual period, although it is possible for a patient to have bleeding from a corpus luteum at the time of flow, unusual but possible.

"The lower midabdomen showed moderate tenderness." That would go perfectly well with appendicitis or with a ruptured cyst, though midabdominal pain is more in favor of ruptured cyst than an appendix.

"Examination of the urine was negative." Although that does not rule out stone in the kidney, it makes me feel it is less likely.

"Examination of the blood showed a white cell count of 17,400." That would be consistent with the temperature and a diagnosis of appendicitis but it also could be due to a ruptured cyst with bleeding in the pelvis.

We also have to consider the possibility of an extrauterine pregnancy, but with an attack five months earlier of a similar nature it would seem unlikely. On the other hand her previous

attack of pain may have been entirely different and she may have had an extrauterine pregnancy which ruptured at this time.

"At operation . . . the appendix was found in the pelvis." That would account for the fact that there was very little tenderness or spasm, only moderate tenderness in the abdomen.

"Scattered over the peritoneal surfaces of the large and small bowel, . . . were flecks of brownish mucoid material." Many times while operating on patients I have seen brownish flecks of material on the parietal peritoneum and on the bowel. It has usually been in the nature of old blood or bloody spots, bluish in character, sometimes purplish, which have been taken to be areas of hemosiderin or ectopic endometrium spilled from a ruptured ovary, or poured through the tube, and growing to the peritoneum or viscera. The fact that this material was mucoid rather confuses the picture for me. It would be my feeling that the appendix might easily be involved in such a lesion.

"This material could be rubbed off the bowel in places . . .", showing that it probably was blood, or lightly attached new growth of a mucoid type. "But in others it seemed to be in a thick layer and did not yield to rubbing." In other words, if there had been a rupture of the ovary five months ago, it might have become organized on the wall of the bowel and therefore could not be rubbed off.

If the appendix was not reddened one would surmise that it was not an acute appendix, but as it is described as nodular and firm it might easily be a lesion in the appendix, either a carcinoid tumor, an adenomyoma or possibly a mucinous tumor of the appendix such as a mucocele or a tumor similar to a pseudomucinous cystadenoma. Mucoceles grow in that region and may give rise to a pseudomucinous peritonitis.

"The right ovary was slightly enlarged but not abnormal otherwise." This spoils my diagnosis, because the ovaries should not have been absolutely normal, and enlargement alone does not answer the question for me. If this ovary had been adherent or had a rupture in it, one would guess that she had a ruptured follicle, or a ruptured corpus luteum, or a ruptured endometrioma or chocolate cyst of the ovary; but as the ovaries are normal it is difficult to make that diagnosis. On the other hand, if this patient was operated upon through an appendix incision and it was necessary to explore the pelvis by mere feeling with the hand and not actually seeing, one might miss a lesion in the wall of the ovary. Most endometriomas of the ovary are adherent because they have ruptured and have become fixed to whatever they happen to lie against after a rupture.

"The left ovary was not seen very well but as nearly as could be determined was normal to palpation." It might be possible that that was the ovary which was in trouble and since it was not seen a lesion might have been missed. Certainly a small bleeding area from a corpus luteum could be missed if one depended on palpation.

"There was a slight amount of turbid fluid in the abdomen." This is rather confusing to me, if it had been bloody fluid I would have thought of a ruptured corpus luteum; if it had been chocolate fluid I would have thought of a ruptured chocolate cyst, but the turbid fluid makes me feel that it must have been inflammatory.

The tubes are not described and whether they are involved in this lesion I cannot answer. I believe the diagnosis is not extrauterine pregnancy because of the history and findings; but since the tubes have not been mentioned, and as we do not know what they were like, we must consider the possibility of extrauterine pregnancy. I feel that this patient probably had a cyst of the ovary which had ruptured probably five months before—if that attack is to be brought into relation with the present one, explaining the old blood in the pelvis and the areas that could not be rubbed off; and then it ruptured a second time producing the flecks that could be rubbed off easily. I believe that she probably had a ruptured endometrioma with a scattering of endometrial-like tissue through the pelvis and an endometrioma of the end of her appendix. Her age, thirty-three, fits in very nicely with endometriosis because it is at about that age that it usually occurs. I will commit myself to the diagnosis of endometriosis, with rupture of an ovarian endometrioma and involvement of the appendix in the tumor.

Clinical Discussion

Dr. Tracy B. Mallory: Dr. Fitchet, this patient was under your care. Perhaps you can tell us a little more exactly what you found at operation and what you think about it.

Dr. Seth M. Fitchet: I congratulate Dr. Meigs on making the diagnosis on something I could not diagnose at operation. This patient did have nausea. The record says she did not. The lower abdomen did reveal moderate tenderness, but it was more marked on the right than on the left; there was distinct spasticity on the right as compared with the left. The record says that the abdomen was soft; it was not soft in the lower half, it was distinctly spastic on the right and less spastic, but still spastic, on the left. Pelvic examination was not done. On rectal examination there were no masses present in either vault, but there was distinct pain and this pain was marked on movement of the cervix.

I did, as Dr. Meigs pointed out, make an improper incision for a pelvic operation. I made an incision for an appendectomy because I be-

lieved from the story and from the physical findings, including temperature and leucocyte count, that this was a fairly typical case of acute appendicitis. So I made the usual right rectus, muscle retracting incision and that accounts for the limited exploration which could be done at operation. The material was mucoid in character and was not bloody. Flecks of this material handled as if it were very viscid mucus. Other particles could not be separated from the bowel peritoneum. There was no bloody material in the pelvis or anywhere else in the abdomen. As nearly as I could describe the free fluid in the abdomen it was turbid and not bloody fluid. The tubes were not mentioned because they were normal, so far as could be determined by feeling them and seeing them through the appendix incision. The right ovary was distinctly larger than the left, not roughened, and it showed no evidence of adhesions; it was freely movable. The left ovary was smaller than the right ovary, but could not be seen so well. As far as it could be seen and felt it was normal. The appendix was shaped very much like one of these telephone dialing pencils, a pencil with a knob on the end of it. The appendix was definitely not injected, but was a fairly long appendix and on the very tip was this ball-mass. We thought of endometriosis and mentioned it, but our findings were not consistent with the diagnosis of endometriosis as we had seen it. We called a pathologist and he was unable to make a diagnosis from the gross appearance of the opened abdomen and the appendix itself. He made a frozen section and was able to report only that it was not malignant. The tip of the appendix was firm and hard. It was not a cystic or mucoid type of swelling; it was definitely hard and firm. The area about the appendix where it lay against the pelvic wall was definitely injected and there was a greater amount of mucoid material there than there was scattered over the other areas involved. In the absence of a sure diagnosis a drain was placed in the pelvic area involved and the wound was closed. She started an apparently normal menstrual flow the day after operation and during the time she flowed from the vagina she had a copious flow from her draining wound, far more than the abdominal findings would lead one to expect. She saturated many dressings daily. This abdominal flow of a bloody-like discharge ceased at the time she stopped flowing from the vagina; it suggested an abdominal wound menstrual flow. Her convalescence proceeded normally and comfortably every other way. Her wound healed promptly without sinus formation.

Dr. Joe V. Meigs' Diagnoses

Ruptured endometrial cyst of the ovary.
Endometriosis of the appendix.

Pathologic Diagnoses

First operation: Endometriosis of appendix. Healing appendicitis.

Second operation: Endometriosis of ovary.

Pathologic Discussion

Dr. Mallory: In corroboration of Dr. Fitchet's statements, the laboratory did see this fluid which was certainly not the ordinary chocolate fluid that one would expect in endometriosis and we did attempt to make a frozen section from the tumor-like swelling of the appendix and were unable to recognize any endometrial glands. When the celloidin sections came through two days later we found frank endometrial cysts in a number of spots along the wall of the appendix, no single one that was very large, but a great many small ones.

Dr. Meigs: If I had known it was mucoid material I do not think I could have made the diagnosis, but the fact that you said it was "brownish" made me feel that it was old blood scattered over the viscera and peritoneum and you do see endometriosis in the appendix not infrequently. Perhaps I would not have made the diagnosis if it had been mucoid alone. The word "brownish" suggested old blood.

Another thing I had difficulty in explaining was the temperature of 101° and the white count of 17,000. That would go all right with a ruptured ovary or corpus luteum but not with an endometrioma, unless it were infected or twisted and had just ruptured.

Dr. Mallory: There was also a certain amount of eosinophilic infiltration of the walls of the appendix, so I think it is well within the limits of possibility that it was appendicitis, but we did not feel perfectly sure that we could differentiate the inflammatory reaction to spilled blood from that attributable to bacterial reaction. In this case I think it is possible that there may be some element of appendicitis.

Dr. Meigs: It would be difficult to explain the symptoms purely on the basis of a chocolate cyst unless it had just ruptured. It gave pain.

Dr. Mallory: There is a little subsequent history that Dr. Fitchet will give us.

Dr. Fitchet: After determining that this was endometrioma the question came up as to what further should be done. Frankly, I did not know. I talked with roentgenologists and I talked with surgeons as to the desirable thing to do. The question to be decided was whether to do a castration by x-ray or a castration surgically. Six months after the appendectomy the girl decided that she preferred surgical castration; so she was readmitted and through a proper incision this time her abdomen was inspected. The enlarged right ovary which had been noted previously was definitely tied

down in the retrocervical position. It was so firmly adherent both to the cervix and posterior structures that it could not be moved without tearing. The process in the ovary was markedly infiltrating the adjacent tissues. The left ovary was not grossly involved. The tubes were not grossly involved. Because we had decided on a surgical castration I removed both ovaries; in removing the right ovary I ruptured a cyst which had not been apparent, and part of the wall of that cyst was firmly adherent to the retrocervical portion of the uterus. I felt that I had not removed all of the involved peritoneum. Following operation she had a normal comfortable convalescence. After consultation with the roentgenologists it was decided to give x-ray therapy because of the infiltrating character of the process. It is now eight months since the second operation and she is symptom free.

DR. MALLORY: At the second operation the endometriosis was, as Dr. Fitchet said, limited to the right ovary and adjacent tissues, where several good-sized chocolate cysts were found. The other ovary contained a corpus luteum but showed no evidence of endometriosis.

DR. MEIGS: Dr. Allen has just asked me why x-ray treatment does not cure them all. As a matter of fact cases that I have seen and advised x-ray treatment have been perfectly satisfactory. I think it ought to work in all cases. We had one case that is worth mentioning. The patient had endometrioma in one ovary which had been removed and later in the other ovary, which was also removed. Then she developed fibroids and began to bleed abnormally. The reason she bled was that in removing the ovaries they did not remove all of the ovarian tissue, just as Dr. Fitchet said he did not think he had. She developed a nodule in the skin and Dr. Vincent removed a piece of this tumor, which turned out to be a typical endometrioma. We then saw her in the Tumor Clinic and this tumor, in spite of the fact that both ovaries had been removed, would swell and cause pain periodically. There were no symptoms of the menopause, no hot flashes and no disturbance of the nervous system. We thought it worth while to treat her with x-ray to destroy the remaining ovarian tissue. Within six weeks she had hot flashes and within six months the lesion in the abdominal wall had atrophied so that it could hardly be felt. Another case had an endometrioma of the rectovesical septum. She had a positive diagnosis made by biopsy and at the end of one year the tumor, which had been the size of a horse-chestnut when x-ray treatment was started, was reduced to a small white scar. I see no reason why others should not be so treated.

CASE 21092

PRESENTATION OF CASE

First admission. A fifty-six year old American housewife entered complaining of intermittent abdominal pain of four months' duration.

Three weeks before entry she first consulted a physician because of an ache which originated somewhere above the left hip and ran down to the hypogastric region, occasionally to the groin. There was no history of nausea, vomiting, chills or fever. A diagnosis of abdominal tumor was made. At the time of admission she was having no abdominal pressure symptoms or any other discomfort.

Her family and marital histories are noncontributory.

Five years before entry she had had what was diagnosed as colitis, and since then she had been on a special diet which had given her some relief. The menopause had occurred six or ten years ago. Since then she had no bleeding but did notice slight leukorrhea. There were no urinary symptoms.

Physical examination showed a well-developed and nourished woman in no pain. The lungs were negative. A slight systolic murmur was heard at the base of the heart. Abdominal examination showed a clearly outlined, firm, freely movable suprapubic mass filling approximately the midportion of the pelvis.

At operation on the third day a large cystic tumor was found filling the entire pelvis and abdomen as far as the umbilicus. There was also a large cystic area on the left side. The omentum and intestines were adherent to the tumor everywhere and it was with some difficulty that these were stripped off the mass. The appendix, which was surrounded by tumor, was freed with some difficulty and removed. The tumor was shelled out of the pelvis and removed. The right ovary was involved, but the left was free of disease. A supravaginal hysterectomy was done, removing the other tube and ovary and part of the broad ligament, which was covered with tumor. There was no evidence of tumor at the end of the operation. There was, however, some bleeding and an area on the small intestine was found oozing rather rapidly. This was sutured.

Her convalescence was uneventful except for a mild colitis consisting of three or four movements a day with a little mucus but no blood. She was discharged on the twentieth postoperative day.

Second Admission. Approximately two and a half years after discharge.

History of Interval. Since discharge she had gained rapidly in strength, her appetite was good and her bowels moved fairly regularly. One month after discharge she received two

x-ray treatments of 1000 R to the anterior and posterior pelvis over a 20 by 20 field.

During the six months before this entry she had four attacks: one six months ago, another three months ago and two within the past three weeks. These attacks were characterized by an aching pain, usually over the left side of the abdomen, and associated with "mucous" diarrhea and vomiting. In addition, she had pain in the left upper quadrant which she described as being similar to labor pains. It came on at any time of the day or night and was relieved by a hypodermic, but not by food or enemas. They were not associated with fever or chills. The last two attacks followed a day in which there was no bowel movement.

On physical examination there was moderate tenderness in the left umbilical region. The liver was felt two centimeters below the costal margin. The right kidney was also felt.

The temperature, pulse and respirations were normal.

Examination of the blood showed a red cell count of 4,540,000, with a hemoglobin of 75 per cent. The white cell count was 6,100, 51 per cent polymorphonuclears. The stools were thick, yellowish brown in color, and contained a large quantity of mucus, some fat, but no blood.

X-ray examination of the abdomen showed kidney outlines on both sides. The left kidney was slightly smaller than the right, but neither was large. There was no evidence of stone. A barium enema was negative. A gastro-intestinal series showed no evidence of obstruction. The coils of ileum were slightly separated and in an unusual position but there were no pressure defects or evidences of extrinsic tumor. An intravenous pyelogram confirmed the previous findings. A Graham test was negative.

Because of the negative studies she was discharged six days after entry without exploration. If further attacks occurred she was to return for operation.

Third Admission. Three weeks later.

Two days after leaving the hospital the patient began to have vague abdominal pains during the middle of the day. In a few hours these pains gradually became more severe and assumed a cyclic periodicity, coming on about every seven minutes. Toward evening they became very severe, knife-like in character, and were sharply localized to an area just above the umbilicus. A physician administered a hypodermic and twelve hours later the pain was so severe that another hypodermic was necessary. The next morning she felt somewhat weak but the pain had disappeared. Seven, five and two days before entry the patient had similar but milder attacks. These attacks had no effect on her bowel habits and were not related to eating. She vomited only at the end of the first severe attack. She felt perfectly well between these attacks. There were no bloody or tarry stools and no diarrhea.

On the third day operation was performed.

Differential Diagnosis

Dr. Lincoln Davis: Dr. Mallory asked me to come down here to discuss this case. I hope he did not have any ulterior motives. I am out of practice at this sort of thing, but I do not believe tumors have changed in the past few years. Treatment may have changed somewhat but the tumors are the same.

The first admission may be briefly dismissed. At operation a large partially cystic tumor was found which undoubtedly originated in the right ovary and was either adherent to or had actually invaded the intestines and the appendix.

"The appendix, which was surrounded by tumor, was freed with some difficulty and removed." I should think that might mean that there was tumor growth on the appendix but the statement is not so clear as one would wish.

"She received two x-ray treatments of 1000 R." What does that mean?

Dr. George W. Holmes: "R" stands for Roentgen, and Roentgen is the term used for specifying x-ray doses.

Dr. Davis: We are probably justified, therefore, in assuming that the tumor was malignant. After a two year interval of good health she develops a new symptom consisting of attacks of pain which are described as "cyclic". There is also a pain described as "similar to labor pains", with a slightly different location. It is not clear to me whether this was really a different type of pain. At the second admission these attacks of pain appeared to be connected with periods of constipation though in the final admission this relationship is denied in the statement "these attacks had no effect on her bowel habits." I wonder if her bowel habits had any effect on the attacks, whether the passage of gas or fecal matter relieved the pain. It does not say.

The record of the abdominal examination is scant. On palpation there was an area of tenderness to the left of the umbilicus. We presume that there was no spasm, no mass, no visible or audible peristalsis. I would feel more comfortable if the vaginal examination were explicitly recorded, but again I assume that it was done and nothing of importance discovered. The patient was not jaundiced; the Graham test was negative. X-ray of the urinary tract was negative and the urine examination presumably showed nothing since it is not mentioned.

We have nothing, therefore, in the way of physical or laboratory findings to back up the story. It seems to me we would really want more data before performing an operation—a view probably shared by her physician since she was discharged without treatment. The

symptoms promptly recurred, however, with increasing frequency and severity and she entered the hospital for the third time.

It seems from this story that the pain was of a colicky sort, a rhythmical colic. It is stated that it was something like labor pains. We could rule out appendix because the appendix had been removed at the previous operation. Gall stones or renal or urethral stones presumably can be ruled out because there was a negative Graham test and a negative x-ray examination. Could this possibly be a uterine colic? We have seen cases of uterine colic due to an extruding fibroid or some other growth in the cavity of the uterus. She had had the body of the uterus removed. The cervix was there, I suppose. A growth in the cervical canal might possibly give rise to colicky pain; but I think that is very unlikely. Intestinal colic due to intermittent small intestine obstruction seems the most likely, possibly a volvulus or adhesions. She had had a previous operation. She had had a growth of some kind which was quite widespread, with adhesions to the abdominal organs and with tumor growth adherent to the tubes and other organs. We know she had x-ray treatment after that, suggesting that there was a question of malignancy. This tumor which she had had removed was very likely an adenocarcinoma of the ovary, one of those malignant growths of the ovary which, after they have broken through the ovary, disseminate all through the abdominal cavity. So that there was every reason for subsequent obstruction, mild obstruction, intermittent obstruction, possibly due to adhesions, to volvulus or to the recurrence of new growth. I should think that the most likely diagnosis was partial obstruction of the small intestine as a result of the conditions which were found at the previous operation.

DR. TRACY B. MALLORY: Dr. Meigs, will you tell us about your findings?

DR. JOE V. MEIGS: This patient had an adenocarcinoma of the ovary, operated on two years and a half ago. She had intermittent attacks of colic-like pain and was studied at this hospital by Dr. Chester Jones, who made a diagnosis of intestinal obstruction involving the small intestine. We did not know what it was due to; there were no physical findings, and the only symptom was occasional colicky pain. When the attacks became more frequent we decided to operate upon her. At operation a tumor of the small intestine was found nine inches from the cecum. The tumor was for the most part submucosal, practically occluding the small intestine, but I believe it probably came in from the outside. We did a resection of the intestine and an anastomosis. She had a stormy convalescence, but is finally out of bed and doing very well. She had no adhesions in the abdominal cavity, and no evidence of tumor anywhere except in the small intestine.

DR. LINCOLN DAVIS'S DIAGNOSES

Adenocarcinoma of the ovary.
Intestinal obstruction.

PATHOLOGIC DIAGNOSES

First Operation: Papillary cystadenocarcinoma of the ovary with invasion to the appendix.
Second Operation: Metastatic papillary adenocarcinoma of the ileum.

PATHOLOGIC DISCUSSION

DR. MALLORY: The specimen which was removed at the first operation was a very large papillary cystadenoma of the ovary. These cysts, with multiple papillary masses sticking into them, we always regard as potentially malignant. In this case, however, histologic examination showed it to be quite rapidly growing, with numerous mitotic figures, and there could be no doubt whatever that it represented established malignancy. Following the first operation she had fairly extensive x-ray treatment, and that is of some interest because certain of these papillary adenocarcinomas of the ovaries are among the group of cancer cases that are most effectively treated by x-ray. In this case, although at the first operation widespread adhesions were present, at the second operation, as Dr. Meigs has told you, only a single tumor nodule was found and that seemed to be actually within the ileum rather than on its outer surface. Interestingly enough it seemed to be at the exact spot where it was noted at the first operation that the intestinal wall was injured in attempting to dig out the primary tumor. From the gross appearance of the intestine at the second operation it would have been impossible to say that it was not a primary carcinoma of the small intestine, although such tumors, of course, are extremely rare. The microscopic sections, however, show very clearly that it is the same type of tumor that was present in the ovary. The first slide is from the primary tumor in the ovary. You can see the large cystic areas with the multiple papillary processes sticking into them. It is a very characteristic type of growth that is approximated in only a few other organs in the body. One might find tumors in the thyroid practically indistinguishable from this and rarely one sees them in the kidney. Primary tumors of the intestinal tract never have a structure similar to this. Here is the section from the resected ileum at the second operation. There is normal mucosa. Then as we go along, the mucosa suddenly becomes replaced by tumor and that tumor has exactly the same characteristics as the original ovarian tumor; so I

think there is no question that we are dealing with a recurrent tumor which arose from a definite implantation at the time of the first operation.

Would you care to hazard a prognosis as to the ultimate outcome in this patient?

DR. MEIGS: Inside the abdominal cavity now there is absolutely no evidence of disease grossly, no lymph nodes, no adhesions, nothing in the mesentery or in the intestines. I should think her prognosis was good.

DR. MALLORY: We certainly have had an occasional case in this group of papillary ovarian cysts that has shown very striking and long continued improvement following x-ray treatment.

DR. DAVIS: Do you remember the Hodenpyle serum?

DR. MEIGS: Yes, it was made from cyst fluid. It is interesting because when we looked up the tumors of the ovary in the hospital and tabulated them, I think there were about 150, we found that the group of tumors that did best after operation were the ones that had the cysts ruptured at the operation. This would be, of course, very heretical teaching, but it falls in line with the serum that Hodenpyle advocated. He advocated treating the patient with the fluid of these tumors.

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PROGRESS IN THE FIELD OF GASTRIC PHYSIOLOGY

REMARKABLE progress in our knowledge of the physiology of the stomach has occurred in the last seven years. The advance was initiated by the original investigations of a Boston worker, Dr. William B. Castle. In 1928, Dr. Castle announced the revolutionary discovery which linked the stomach with the bone marrow and gave a simple physiological explanation for the etiology of pernicious anemia. Subsequent research by Dr. Castle and his co-workers[1] established the fact that the stomach secretes a factor, probably an enzyme, which reacts with certain constituents of the food closely associated with vitamin B_2 (G)[2], to form material necessary for normal blood formation. In pernicious anemia this "intrinsic factor" is absent or greatly diminished and a conditioned deficiency disease exists in spite of an adequate diet containing the necessary "extrinsic factor". The "intrinsic factor" was shown to be absent in saliva and in the duodenal secretion. It was dissociated from pepsin, hydrochloric acid, and rennin. It reacted with the "extrinsic factor" at neutrality and was thermolabile.

Knowledge was further advanced by the work of Sturgis and Isaacs, who showed that whole pig stomach was a potent source of material effective in alleviating pernicious anemia. It is believed that the "intrinsic factor" present in the mucosa of the pig's stomach reacts with the "extrinsic factor" present in the preparations to form the active principle.

Recently Meulengracht and his coworkers in Copenhagen[3, 4, 5] have added anatomical knowledge to this subject. They have demonstrated that, topographically, the glands of the cardia, fundus and pylorus are remarkably well separated in the pig's stomach, which is very similar in this respect to the human stomach. Pepsin, hydrochloric acid and rennin were secreted chiefly by the fundus glands. Preparations from the whole wall of the pig's pylorus, however, were active in pernicious anemia, whereas preparations from the fundus were inactive. The activity of preparations from the cardia is still uncertain. Failure to take into consideration the anatomical distribution of the different types of glands has resulted in the contradictory and uncertain findings of previous workers in this field.

A practical corollary of the recent investigations is that the presence of the stomach, in particular the pyloric region of the stomach, is necessary to health, and that, when subtotal gastrectomy is performed, it would be wise to leave the pyloric glands intact.

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HOUSE BILL 756

ONE of the important bills before the Massachusetts Legislature this year, House 756, imposes a definite responsibility on the people of the Commonwealth because of its significance as a public health measure. It is designed to provide an approved quality of service by the medical profession through an amendment to existing laws governing the registration of physicians.

The Board of Registration in Medicine is now obliged to examine all applicants for licensure as physicians, who have had a premedical education equivalent to that required for graduation from a high school, and who are graduates of a chartered medical school which gives a full four years' course of instruction of thirty-two weeks in each year. The specified premedical experience is so indefinite that the adequacy of the intellectual preparation for study of the many scientific fields included in medicine is open to question. The specified requirement of graduation from a medical school which exists in the present law is also lacking in important particulars, because a school would simply have to show that its doors were open for thirty-two weeks of a full four years' course, and that exercises were continuously carried on for these periods. As to the scope or quality of instruction, no state official has any power to determine whether a given school is worthy of recognition according to generally accepted standards. All states of the Union, except Massachusetts, have been engaged in requiring medical schools to raise the quality of curricula or retire from the field.

For many years petitions have been presented to our legislature for changes, in conformity with those taking place throughout the Union, which would give to the Massachusetts Board of Registration in Medicine authority to accept for examination graduates of approved medical schools only. Hitherto this authority has been withheld, and the graduates of any school with the elementary requirements now in force must be recognized by the Massachusetts Board. Even a cult which teaches medicine for the time specified in the law, stands on an equal footing with the highest grade medical schools, so far as the state is concerned.

Massachusetts has seemed to be quite indifferent, respecting the quality of medical practice within her borders, and unconcerned about her standing with the other states. These attitudes are disconcerting, for with the advances in medicine every state should be keenly alive to the wisdom of having the people provided with the best possible preventive and therapeutic resources. The refusal of other states to enter into reciprocal recognition with the Massachusetts Board of Registration is irritating, to say the least. Even though these matters have been repeatedly explained, support of the effort of the Committee on Legislation by the medical profession has been meager.

If the five thousand members of the Massachusetts Medical Society would engage in a wide and well-planned educational campaign designed to reach the intelligent groups, the legislature would, we believe, give to the proper officials authority to demand of medical schools standards of education approved by the Council on Medical Education and Hospitals. The humiliating situation can only be relieved by the legislature.

The hearing on bill 756 will be held March 7, Room 480, State House, Boston at 10:30 A.M., before the Committee on Education. Meantime, see your Representative and Senator and get others to assist. Attend the hearing if possible.

THE REGISTRATION OF CHIROPRACTORS

A PETITION for the registration of chiropractors (House 1157) is before the Massachusetts legislature, and the Committee on Public Health will conduct a hearing on this appeal March 7 at the State House. This Commonwealth through its legislature, and its people by a referendum, have heretofore declined to recognize this cult.

The wish to secure recognition of this cult in Massachusetts is so insistent on the part of these practitioners, that this new attempt must be met by an appeal to the intelligence of the legislators. This must be directed by the medical profession, and the attitude of the great majority who voted against recognition of these practitioners must be emphasized at the hearing. The claims of chiropractors for efficiency in dealing with diseased conditions have been analyzed by physicians and found wanting.

Massachusetts has adopted the policy of insisting that all who practice the healing art must meet common requirements which are, in substance, graduation from a chartered medical school which gives a full four years' course of instruction of thirty-two weeks in each year, and also passing the examinations of the Board of Registration in Medicine.

We have not been informed of the existence of a chiropractic school which meets these requirements, and the only way for these people to secure registration under present conditions is to have a board in sympathy with their wishes. Chiropractors practice medicine, and should be possessed of sufficient knowledge to meet the problems of disease. Diagnosis is the essential factor in dealing with disease, and requires a well-trained mind and ability to use and interpret the many technical and scientific demonstrations by which one may know what is taking place in the human body, but chiropractors claim to depend very largely on palpation of the spine for indications of treatment.

Medicine is dedicated to the cure and prevention of disease and must, logically, endeavor to protect the people against incompetence in every form. If physicians perform their duty, men of influence must be present at the hearing, March 7, to show the Committee the right course to pursue in dealing with this petition.

THE RECOGNITION OF MAGNETIC HEALERS

ARSENE J. PARE is the petitioner for the establishment of a board of examination and registration to regulate the practice of magnetic healers as appears in House Bill 1458. This has been assigned to the Committee on State Administration and the hearing will be held March 8. (Room 423, State House at 10:00 A.M.)

Under the original Act creating the Board of Registration in Medicine, magnetic healers were exempted from the operation of the law except that the practitioners of the exempted classes may not practice or attempt to practice medicine. This bill confers the right to practice medicine.

Magnetic healing has never occupied a prominent position in the healing art in this section. According to the bill, members of the board must be "gifted with magnetic healing" and an applicant for registration must be "capable of examining nerve conditions by his magnetic power". The examination of applicants for registration "shall include the subject of nerves". Registered magnetic healers shall comply with the rules and regulations governing the reporting of contagious diseases and deaths. Magnetic healing is defined in this bill as "the science of reviving and producing life and circulation in the nerve system and cells, so as to heal all nerve affections". In the first instance the Governor must, in the selection of members of this board of examiners in magnetic healing, determine whether such appointees are "gifted with magnetic healing". Even with his recognized ability, the acumen of His Excellency will be put to a severe test. A definite danger to the public health policies of the State exists in giving to any person, not a physician, the responsibility of dealing with communicable diseases and reporting of deaths.

The definition of magnetic healing in this bill is extremely vague and carries the assumption that practitioners of this cult have an ability beyond that of physicians.

With the concentration of well-equipped minds on the best methods of treating disease, there seems to be no likelihood that magnetic healing will contribute any better methods of relieving suffering than those now used by the medical profession.

This is another vicious attempt, on the part of untrained practitioners, to invade the field of medical practice.

The legislature ought to give the petitioner leave to withdraw his bill.

Doctors should be present at this hearing.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

WAITE, J. HERBERT. M.D. Harvard University Medical School 1916. Clinical Professor of Ophthalmology, Harvard University Medical School. Ophthalmologist, New England Deaconess Hospital. Address: 7 Bay State Road, Boston, Mass. Associated with him is

BEETHAM, WILLIAM P. M.D. Harvard University Medical School 1926. Assistant in Ophthalmology, Harvard Medical School. Assistant Surgeon, Massachusetts Eye and Ear Infirmary. Ophthalmologist to the New England Deaconess Hospital, New England Baptist Hospital, Boston Lying-In Hospital and Lakeville State Tuberculosis Sanatorium. Address: 7 Bay State Road, Boston, Mass. Their subject is "The Visual Mechanism in Diabetes Mellitus." Page 367.

FLOYD, CLEAVELAND. M.D. Harvard University Medical School 1903. Physician-in-Chief, Boston Health Department, Division of Tuberculosis. Consultant, Boston Lying-In Hospital. His subject is "Pulmonary Tuberculosis and Pregnancy." Page 379. Address: 246 Marlborough Street, Boston, Mass.

SCHALL, LEROY A. M.D. Jefferson Medical College of Philadelphia 1917. F.A.C.S. Instructor in Laryngology, Harvard University Medical School. Associate Surgeon, Massachusetts Eye and Ear Infirmary and Massachusetts General Hospital. Assistant Laryngologist, Collis P. Huntington Memorial, Palmer Memorial and Robert Breck Brigham Hospitals. His subject is "A Modified Tracheotomy Tube." Page 386. Address: 270 Commonwealth Avenue, Boston, Mass.

EPSTEIN, ALBERT A. B.S., M.D. New York University and Bellevue Hospital Medical College 1905. Attending Physician, Beth Israel Hospital and Hospital for Joint Diseases, New York City. His subject is "The Nephritides." Page 387. Address: 70 East 83rd Street, New York City.

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY*

THOMAS ALMY, M.D.,
Chairman,
140 Rock Street,
Fall River, Mass.

C. J. KICKHAM, M.D.,
Secretary,
524 Commonwealth Avenue,
Boston, Mass.

TREATMENT OF TOXEMIAS OF LATE PREGNANCY

The only absolute cure of toxemia of pregnancy is the removal of the fetus from the mother's body. As not all toxemias are equally severe, however, and some apparent toxemias are in reality true nephritis, there is a definite place for medical treatment.

*A series of short selected articles by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

Consideration of the treatment of toxemias may be considered under the headings of prophylaxis, toxemic stage and eclampsia.

Prophylaxis implies adequate prenatal care, with advice as to diet, exercise and proper elimination. Routine antepartum examinations, at biweekly intervals during the last trimester of pregnancy, should detect the majority of toxemias in their incipient stage, although occasional fulminating cases will occur.

Toxemic stage. When a blood pressure of over 140/80 or more than a very slight trace of albumin is detected, immediate hospitalization of the patient is called for. Rest in bed, low protein, salt free diet, mild catharsis, and sufficient sedatives are necessary. Fluids should be forced unless there is marked edema. Four hour blood pressures and daily urinalyses are essential.

Mention must be made of the so-called "dehydration treatment", which limits the amount of fluid intake to the amount of urinary excretion. Sufficient trial of this method has not yet been made for a proper evaluation.

Plasmapheresis, washing the blood corpuscles free from toxines in the serum, is too complicated for use except in specially equipped hospitals.

The use of magnesium sulphate intravenously has been extensively advocated. Its proponents claim not only the sedative effect of the salt on the nervous system, but that it also causes resorption of fluid from the tissues into the circulation, resulting in increased elimination. Twenty cc. of a ten per cent solution may be given and repeated at six to eight hour intervals.

So long as blood pressure does not rise or the albumin increase in amount, medical treatment may be continued. A rise in systolic blood pressure to 160 degrees or the increase of albumin to 1/4 of 1 per cent, severe headache, blurring of vision or epigastric pain demand termination of the pregnancy. The method to be employed should depend upon the mechanical conditions present.

In a multipara with previous normal labors, or in a primipara under thirty with good pelvic measurements where no mechanical difficulty in delivery is to be expected, artificial rupture of the membranes, with or without the insertion of a bougie (a sterile rectal tube makes an excellent bougie) is usually sufficient. The Voorhees bag may be used, but is accompanied by an increased risk of infection. Labor having been induced, the patient should be kept well under the influence of barbiturates and a "hands off" policy maintained with regard to operative interference and anesthesia.

In a primipara over thirty with an unengaged presenting part and a long undilated cervix, and especially if the pelvic measurements are contracted, a long labor and difficult delivery may be anticipated. In these patients, cesarean is usually the method of choice. Theoretically, either local or spinal anesthesia should be ideal in these cases. The dangers of spinal anesthesia in cesarean, and especially with hypertension, and the general unsatisfactoriness of local anesthesia, make well-given gas-oxygen safer in the long run.

The after-treatment consists chiefly in resuming the previous medical treatment. Fluids may be given by mouth, hypodermoclysis or intravenously. Only water, fruit juices and milk should be given by mouth during the first seventy-two hours.

Eclampsia. The majority of obstetricians now favor some modification of the Stroganoff régime with or without induction of labor. Cesarean section should not be done except in the presence of a definite mechanical indication.

The original Stroganoff method consisted in putting the patient to bed in a darkened room, plugging the ears and avoiding handling as much as possible. Morphia, gr. 1/4 on entrance and repeated three hours later. One hour after entrance chloral hydrate grs. xxx by rectum and repeated every six hours until convulsions cease.

Because of the depressing effect of morphia and chloral, many obstetricians now favor the use of barbiturates. Sodium amytal, grs. xv may be given by mouth, or if unconscious by rectum, and repeated in grs. vi doses as often as necessary to keep the patient well narcotized. The narcosis is continued until after convulsions cease. If there is acidosis, 500 cc. of ten per cent glucose should be given intravenously, accompanied by thirty units of insulin. As the patient improves, the same medical treatment already laid down for toxemias should be followed.

MASSACHUSETTS LEGISLATIVE NOTES

THE BILLS RELATING TO VACCINATION

House bill 60, which was the subject of a hearing before the Committee on Public Health February 19, was ably supported by the Department of Public Health; by Dr. S. B. Woodward; the Massachusetts Medical Society as represented by the President, Dr. Robey, and the Treasurer, Dr. Butler; Dr. M. J. Rosenau of the Harvard Medical School, and Dr. C. M. Hilliard, Professor of Biology and Public Health, of Simmons College.

Two members of the legislature also advocated the passage of this bill which is designed to extend the requirements of vaccination to pupils in the private schools of this Commonwealth. Mrs. Henderson again headed the opposition to this measure by arguments against the invasion of the person in the use of an alleged dangerous vaccine, and by assertions that a large number of deaths and serious disabili-

ties are caused by this procedure. She presented several witnesses who reported deaths and serious illness alleged to be due to vaccination.

In addition to quotations from many people who are opposed to vaccination, she made use of the Fitchburg epidemic in support of her arguments and made statements which were shown to be false by Dr. Chadwick, Commissioner of Health. She apparently believed that any illness following vaccination must be the result of this treatment. Her address seemed to tire the Committee, but she was persistent in repetitions and sensational illustration of her beliefs and was permitted to consume a large part of the time.

She championed the opposition bills, **House 623** designed to eliminate all of the vaccination requirements, and **House 755** which requires that physicians guarantee the purity of the virus to be used. All of her arguments were combated effectively.

The most encouraging feature of the hearing was the comparatively small attendance of the opponents of compulsory vaccination.

As we go to press, notification has been received that the following bills have been given leave to withdraw:

H. 623. Bill making vaccination voluntary.

H. 755. Bill to prevent vaccination or inoculation with impure virus or serum without consent.

The following bill has been reported favorably in House. Referred to Ways and Means.

H. 758. Resolve providing for an investigation by a special unpaid commission of the Public Health laws and practices of Public Health in the Commonwealth.

SCHEDULE OF HEARINGS

Thursday, **March 7,** at 10:30 A.M., in Room 480, State House, before the Committee on Education.

H. 756—A petition of the Massachusetts Medical Society for legislation relative to the qualifications of applicants for registration as physicians.

Approved by the Committee on State and National Legislation of the Massachusetts Medical Society.

Thursday, **March 7,** at 10:30 A.M., in Room 450, State House, before the Committee on Public Health.

H. 1157—A petition of Henry J. Kennedy for establishment of a board of examination and registration to regulate the practice of chiropractic.

Opposed by the Committee.

Friday, **March 8,** at 10:00 A.M., in Room 423, State House, before the Committee on State Administration.

H. 1458—A bill to create a board of examination and registration to regulate the practice of magnetic healers.

Opposed by the Committee.

MISCELLANY

NOMINATIONS BY THE GOVERNOR

His Excellency, Governor Curley, has nominated three trustees of the Massachusetts General Hospital for consideration of the Executive Council. They are Mr. Joseph A. Tomasello, Treasurer of the Democratic State Committee; Mr. Edward A. Filene, a business executive, who has shown interest in sociology, and Mr. Henry V. Morgan, Treasurer of the Boston Bridge Works, Inc.

These nominations will, if approved by the Council, take the places vacated by Mr. Edwin S. Webster, former Mayor Andrew J. Peters, and Mr. Joseph H. O'Neil who died recently. Miss Dumaine of Groton was reappointed a trustee of the Hospital.

Other appointments, by the Governor, of interest to the medical profession, are Eva M. Watson, trustee of the Medfield State Hospital; Edna W. Dreyfus reappointed trustee of the Boston State Hospital, and Rose Herbert, trustee of the Grafton State Hospital.

THE APPOINTMENTS OF THE DEAN OF THE HARVARD MEDICAL SCHOOL, AND DEAN OF THE SCHOOL OF PUBLIC HEALTH

The Board of Overseers of Harvard University has approved the appointment of Professor Charles Sidney Burwell as Dean of the Harvard Medical School and also that of Professor Cecil Kent Drinker as Dean of the School of Public Health.

Dr. Burwell was born in 1893 and graduated in medicine from the Harvard Medical School in 1919. He is Professor of Medicine at the Medical School of Vanderbilt University, Nashville, Tenn. He will fill the position made vacant by the resignation of Dean David L. Edsall.

Dr. Drinker was instructor in physiology at the Harvard School from 1916 to 1918 and has been Professor of Physiology since then. He has also served as Assistant Dean of the School of Public Health.

CORRESPONDENCE

HOUSE BILL 340 AND ITS DANGERS

Editor, *New England Journal of Medicine,*

House Bill 340 proposes in effect to exclude insurance companies from any function in the administration of the Workmen's Compensation Act and to substitute a monopolistic "State Fund." Passage of this bill as it stands would be a calamity.

One of the greatest benefits to the workman under the Compensation Act is the assurance of proper medical care. After all, if a workman gets seriously smashed up, it is the surgeon and the surgeon alone who may make him whole. And if for any reason he fails to get proper surgical treatment he falls into bad luck which no 500 weeks of compensation can make up to him.

Many years ago some few surgeons interested themselves in the problems of repair of those crippled in industry. At first there was no compensa-

tion—no compensation for the surgeon, certainly, and no way of assuring proper hospital treatment and care in convalescence. But some of us kept at it as best we could.

Then came the Workmen's Compensation law and oddly enough (just as in the proposal now under consideration) the first draft entirely neglected the whole medical problem. This is almost unbelievable, but true.

Very soon the late Judge James Carroll, father of the law, appreciated the situation and acted.

The writer happened to be the man he selected to arrange for open meetings at the State House to get the doctors into line and to head a committee of medical men to act as medical advisers to the board.

This committee served (unpaid) for years and did a great deal of work. Almost all of the changes in the law by later amendment, that concerned medical matters, originated with this committee, and almost all of the forms of medical procedure were from the same source.

The writer worked in the early days and ever since with the board, with the insurance companies and against them, and devoted much professional effort at the City Hospital, in the army before and after the war, and in private practice to what we now call bone and joint surgery and the surgery of reconstruction, with not inconsiderable contributions to new techniques and methods, and to the testing and use of known ways of treatment.

So some have seen the game through up to the present time.

And for twenty years and more it has been a matter of education of insurers and employees—and the board—of slow working out of methods of coöperation of the board, the insurers and the doctors, in order to get results.

And we have gotten results.

So that to-day the injured workman is assured of competent treatment, and of especially expert treatment when that is needed.

The best surgeons have found it possible to coöperate in this accomplishment.

How wide the coöperation has been, appears from the list of eleven hundred doctors in Massachusetts that have been paid by one company.

And the results compare most favorably with those of any state—are better, we think, than those under state fund administration.

It would be a great pity to waste all this painfully won accomplishment of years.

And, make no mistake, if the insurance companies are excluded from the operation of this act, the whole thing is scrapped, and we go back to a medical chaos like that of 1913, to work the scheme out again, and likely enough to work it out worse instead of better than we now have it, to say nothing of the long complicated mess of the readjustment period.

And what is there to be gained?

A case might perhaps be made out for a competitive state fund, but not for a monopolistic one, in the opinion of very many of the medical profession.

Those who know the situation best, those who view it most broadly, who see it in terms of salvage of human efficiency, of return of the damaged workman to his previous self, who believe that the doctor's best service is the best recompense the workman can have, are concerned over a situation that seems to jeopardize what has been so hardly won.

No human thing is perfect or beyond improvement, but this bill as it stands should not pass.

Every doctor should try and bring about an understanding by his legal representatives that the medical profession is against it, not only in the interest of the doctor, but even more in the interest of the injured man whom we try to restore to health.

F. J. Cotton, M.D.

DID THE MODEL FOR LEONARDO'S "MONA LISA" HAVE A LEFT SUPRA-ORBITAL DERMOID?

Mr. Editor:

In studying a reproduction from a copy of this most celebrated of portraits, your correspondent has been struck with the appearance just above the left fronto-temporal angle of the portrait.

If one observes this spot carefully, one gets the effect through the shading, of a small soft oval, somewhat ill-defined swelling just above and to the outer side of the left eyebrow, extending to the junction of the hair line. It suggests a small dermoid cyst at this site.

Perhaps some of your readers, interested in art, after looking at a copy of the picture with this in mind, may be reminded of certain similar cases they have seen or operated upon.

Very truly yours,

Wm. Pearce Coues, M.D.

AN EXPLANATION OF THE DOSAGE OF DILAUDID COMPOUND TABLETS

February 20, 1935.

Editor, *New England Journal of Medicine*,

Our attention has been called to page 316 in the *February 14 issue* of the *Journal*, on which is reprinted a letter from Dr. Paul Nicholas Leech, Secretary of the Council on Pharmacy and Chemistry of the A. M. A., listing products recently accepted. This list includes a dosage form of Dilaudid which is here given as "*Dilaudid Compound Tablets ½ grain.*"

We believe that this listing is likely to cause some confusion in prescribing Dilaudid, since these tablets are not a mixture of Dilaudid with other drugs, but a *Compounding* tablet used to facilitate the preparation of prescriptions. Frequently, Dilaudid is prescribed in the proportion of ½ grain in 4 ounces of vehicle and then given in teaspoonful doses, each of which contains 1/64 grain.

We should like to correct any impression that Dilaudid can ordinarily be given in ½ grain doses. A ½ grain dose of Dilaudid is considered as equivalent

to 2½ grains of morphine, and this would be likely to cause depressive symptoms in a patient who has not developed a tolerance to opiates.

We have taken every precaution to prevent these tablets from being dispensed as such, and have labeled them "Dilaudid Compounding Tablets ½ grain for compounding only. Do not dispense."

We trust that you understand our position in this matter, and hope that you can call the attention of your readers to the correct use of Dilaudid Compounding Tablets so that any accident which might result from the administration of such a large dose may be prevented.

Very truly yours,

BILHUBER-KNOLL CORP.,
F. B. WESTERN, M.D.

154 Ogden Avenue,
Jersey City, N. J.

DOCTORS' DEALINGS WITH LAWYERS

The Law Society of Massachusetts

February 13, 1935.

Editor, *New England Journal of Medicine*,

A communication in your issue of February 7, 1935 from Dr. W. A. Hutton has been called to my attention.

The subject of the doctor's dealings with lawyers in accident cases was dealt with by me in an address delivered at the Phi Delta Epsilon Fraternity recently. I made a fairly careful study of the situation and, for their future guidance, drew a sample letter which I distributed to the doctors attending this meeting.

If I may be permitted an opinion, I would like to say that Dr. Hutton is a little too hard on the lawyers generally, and his statement on the law with regard to a doctor's right to collect for appearing as a witness is erroneous.

I have done business with hundreds of physicians during the time I have been in practice and have never yet found it necessary to have more than a friendly talk with a physician when the fund available was not large enough to pay his entire bill, and my entire fee. A personal friendly talk, it is suggested, is all that is necessary for men of two honorable professions to adjust their differences to their mutual satisfaction.

If the Medical Societies would invite the officers or a representative of the County or City Bar Association to come before their Body and explain to them their full rights and give them a form letter to guide them in their dealings, the medical men would do themselves a service, encourage friendship between our respective noble professions, and make unnecessary the acrimonious epistles of Dr. Hutton and others.

If you deem it advisable, you have my permission to set out the form letter and the citation of authorities which I have annexed hereto for the benefit of your readers, and let me say as an officer of the Law Society of Massachusetts that our organization stands for mutual aid and coöperation between the medical and legal professions, and would censure most severely any lawyer against whom a just complaint by a doctor could be proved.

Very respectfully yours,

JOSEPH SCHNEIDER,
Secretary of The Law Society of Massachusetts.

SAMPLE LETTER

Dear Lawyer:

I am treating your client and my patient John Jones for the injuries sustained by him in the accident of June 1, 1934 at Summer and Washington Streets, Boston. I am willing to attend Court without summons if you will give me an adequate telephone notice in advance of the time you may call me. Mr. Jones has agreed that he is to pay me in any event for all professional services rendered no matter what the outcome of his claim for damages may be, but that if any moneys come into your possession on account of his injuries and he has not yet paid me, you are to retain and forward to me sufficient to pay the charges for my medical services and a reasonable fee in addition for my Court attendance as an expert if I am called to testify.

I would appreciate an acknowledgment for my files of your assent to this arrangement. Kindly advise me in what Court you start suit and the status of the case from time to time, and oblige.

Very respectfully yours,
(DOCTOR.)

Dear Lawyer:

The foregoing which I have read is agreeable to me and you are authorized to pay in my behalf in accordance with the above understanding.

(PATIENT-CLIENT.)

Reference:

1. Lawyer has no right to pay doctor unless authorized.

Falardeau v. Washburn, 199 Mass. 363.

2. Doctor has no right to be paid for Court attendance unless there is an express agreement to pay and he comes without summons or he is called as an expert.

Barrus v. Phaneuf, 166 Mass. 123.

Keown & McEvoy, Inc. v. Verlin, 253 Mass. 377.

3. An agreement to share in the fruits of the litigation or take a percentage thereof or to look only to the outcome of the litigation for payment of a doctor's bill is void as against public policy.

Weinberg v. Magid, 1934 Mass. Ad. Sh. Page 155.

RECENT DEATHS

KARCHER—EDWARD WINSLOW KARCHER, M.D., of Lynn, Massachusetts, with an office at 416 Marlborough Street, Boston, died at his home, February 18, 1935. He was born in Newton Centre, Massachusetts, in 1882, and graduated in medicine from the Middlesex College of Medicine and Surgery in 1916.

He was a Fellow of the Massachusetts Medical

Society and the American Medical Association, and a member of the Tedesco Club.

His widow, Mrs. Ada (Quincy) Karcher, three sisters and a brother survive him.

SIMPSON — James Edwin Simpson, M.D., of 26 Chestnut Street, Salem, Massachusetts, died in that city, January 19, 1935. He was born in 1869, and graduated from the Harvard Medical School in 1891.

He was a Fellow of the Massachusetts Medical Society and the American Medical Association.

WHITNEY—Edward William Whitney, M.D., Superintendent of the Northampton State Hospital, died February 16, 1935. Previous to his appointment as Superintendent in 1933, he had served the hospital for twenty-five years as assistant physician and twice as acting Superintendent. He was also Assistant to the Commissioner of Mental Diseases of Massachusetts.

Dr. Whitney was born in Putnam, Conn., in 1879, and graduated from the Harvard Medical School in 1903 and was an interne at the Boston City Hospital.

He was a Fellow of the Massachusetts Medical Society, the American Medical Association, and a member of the Massachusetts Psychiatric Society, the American Psychiatric Society, and the New England Psychiatric Society.

His widow, Dr. Harriet Wiley Whitney, assistant physician at the Northampton Hospital, survives him.

BRAGDON—Horace Elwood Bragdon, M.D., of 77 Bartlett Road, Winthrop, and former head of the Chelsea Memorial Hospital, died at his home, February 17, 1935. He was born in East Boston in 1867, the son of Captain Byron F., and Angie Elwood Bragdon.

After graduating from the Harvard Medical School in 1891 he served as interne at the Boston City Hospital and later studied in Vienna. He was a Fellow of the Massachusetts Medical Society and the American Medical Association and a member of the Harvard Club, Temple Lodge of Masons and Zenith Lodge of Odd Fellows.

He was Vice President of the Sumner Savings Bank, Director of the Columbia Trust Company and the Enterprise Coöperative Bank.

His daughter, Mrs. John Gore, of Canajoharie, N. Y., and two grandchildren survive him.

BRADFORD—Cary Carpenter Bradford, M.D., of Southbridge, Mass., died at Woodstock, Conn., October 20, 1934. He was born in 1855, graduated from the Harvard Medical School in 1882 and was a former Fellow of the Massachusetts Medical Society. He retired from practice several years ago.

NOTICES

REMOVAL

William D. Rowland, M.D., announces the removal of his office to 84 Commonwealth Avenue, Boston.

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, March 7, in the Amphitheatre of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students. These clinics will be repeated on Thursdays until May.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian. These are open to all physicians.

BOSTON UNIVERSITY SCHOOL OF MEDICINE SURGICAL CLINIC AT THE BOSTON CITY HOSPITAL

Friday, March 1, 12-1, Cheever Amphitheatre.

Dr. Augustus Riley, Assistant Professor of Genito-Urinary Surgery, Harvard Medical School, and Visiting Surgeon, Boston City Hospital, will discuss "Surgical Lesions of the Genito-Urinary Tract."

Physicians and medical students are invited.

REPORTS AND NOTICES OF MEETINGS

SUFFOLK DISTRICT MEDICAL SOCIETY

A joint meeting of the Suffolk District Medical Society and the Boston Medical Library was held at the Boston Medical Library on the evening of January 23. Dr. Chester S. Keefer was the first speaker and discussed "The Differential Diagnosis of Degenerative Arthritis." The term "degenerative arthritis" is synonymous with hypertrophic arthritis, osteo-arthritis, and arthritis deformans. The pathological picture of this condition consists of a fibrillation of the cartilage, compression of the underlying subchondral tissue, a loss of cartilage with an attempt at repair and the formation of exostoses. In the spine there is a disorganization of the discs with prolapse and compression with exostosis formation. Degenerative changes in the joints normally increase with age and tend to appear in the following order: knee, acromioclavicular joint, elbow, hip, metatarsal, and spine. The chief predisposing or accelerating factors are first, advancing age; secondly, certain occupations as the use of compressed air hammers; thirdly, gross trauma as a fracture involving the joint surface; fourthly, postural defects; fifthly, excessive weight; sixthly, previous damage by infection, especially typhoid of the hip; and seventhly, the loss of pain and position sense. Dr. Keefer then considered in detail the special accelerating factors in each of several joints.

In discussing special forms of degenerative arthritis, Dr. Keefer first spoke on Charcot's joints which may occur in tabes dorsalis where there is an extreme degenerative change with the loss of pain

sense. They also occur in syringomyelia and in certain peripheral nerve diseases. In hemophilia, trauma causes hemorrhage into the joint and into the bone which leads to marked degeneration and cystic formation within the cancellous bone. There is probably no relation between arthritis and alkaptonuria.

Degenerative arthritis must be differentiated from diseases in the neighboring bone, functional conditions such as strain, and new processes superimposed upon the arthritis. As for diseases that may occur in the neighboring bone, one must exclude in the case of the spine: Marie-Strümpell's disease which rarely occurs after forty years of age, Charcot's disease, hyperparathyroidism, Paget's disease, cord tumors, posterior dislocation of the intervertebral discs, tumors of the vertebrae either primary or secondary, infections of the vertebrae, collapse of a vertebra, and aneurysmal destruction of the vertebrae. Conditions which may be superimposed upon degenerative arthritis are infections, proliferative joint disease, gout, and, rarely, tumors. In the spine, the pain may be exaggerated by coughing, sneezing, or defecation and often has a radicular distribution. There may be areas of anesthesia and a limitation of motion.

In arthritis of the elbow of this type there are frequently loose bodies which may cause the joint to lock. The knee becomes stiff, crepitates on motion, and flexion is limited. When the synovial membrane is pinched, infarction occurs with a hemorrhagic effusion and loose bodies are formed. In summary, degenerative arthritis normally increases with age, often creates lesions that do not cause pain, and when symptoms do arise, the condition must be distinguished from neighboring bone lesions or superimposed conditions.

Dr. Walter Bauer then spoke on "The Differential Diagnosis of Rheumatoid Arthritis." He presented a simple classification of those cases where the origin is known and where it is unknown. There are several subdivisions of the known origin group, chief among which are first, traumatic as in sprains, etc.; secondly, bacterial including the tubercle bacillus, the gonococcus, staphlyococcus, etc.; thirdly, the neuro-arthropathies (Charcot's) including tabes, syringomyelia, and leprosy; fourthly, metabolic as gout; fifthly, constitutional, as hemophilia and hysteria; and sixthly, anaphylactic as in serum sickness.

In the group where the origin is unknown or uncertain there are degenerative arthritis, proliferative arthritis, rheumatic fever and others. Rheumatoid arthritis, synonymous with atrophic and proliferative arthritis, is a chronic disease of unknown etiology characterized by relapses. The nature of the onset tends to vary considerably, and the periods between relapses become shorter and shorter until the patient assumes a steady downhill course. Dr. Bauer stressed the fact that rheumatoid arthritis can affect any age and is quite common past middle age. Constitutional symptoms are usually present, are often marked, and frequently precede joint symptoms. The small joints are most often affected, frequently bilaterally and symmetrically. The first complaint of these patients is sometimes that associated with paresthesias. There is usually marked muscle weakness and there are definite vasomotor symptoms with cold, perspiring extremities. The skin is shiny and atrophic and may have pigmentary changes. The temperature, white count, and sedimentation rate are all increased demonstrating this disease to be a general, constitutional condition.

The joint changes are all secondary to a proliferation of the synovial membrane which extends as a pannus formation into the joint space. This pannus erodes the articular cartilage and causes fibrous tissue ankylosis. Occasionally there is a subchondral proliferation of the connective tissue so that there is destruction of the cartilage from below as well as from above.

Rather than take up all of the conditions with which rheumatoid arthritis may be confused, Dr. Bauer chose to consider a few case histories in which mistakes had been made. The first of these concerned a nineteen year old girl who had developed a painful knee joint after a previous upper respiratory infection. This developed into a migratory arthritis requiring opium for the relief of pain. After a positive complement fixation test and the gonococcus had been found in the aspirated joint fluid, the diagnosis of gonorrheal arthritis was made. The features of this case are the onset like rheumatic fever, the fact that the complement fixation test gave the first definite clue, and it shows the value of diagnostic aspirations.

The second case was also one of gonorrheal arthritis in a patient where all smears as well as the complement fixation test were negative. The first clue was the admission of the husband that he had been previously infected, and the diagnosis was established when the smears became positive after a provocative dose of gonococcus vaccine. With regard to gonorrheal arthritis it is necessary to suspect all cases; the clinical manifestations may be indistinguishable from those of rheumatoid arthritis; it may occur days or years after the infection; there may be recurrences; and the onset is often acute with chills and a moderate fever. There is a leucocytosis, a migratory polyarthritis, and it usually affects the large joints. Repeated smears are necessary in the search for the organisms; the complement fixation test is valuable, being positive in eighty per cent of the cases; diagnostic taps often help; provocative doses of vaccine are a distinct aid; and x-rays must be read with caution because of the rapid bone atrophy. The prognosis is very good, there being a sixty to seventy per cent chance of cure.

A nineteen year old boy entered with the diagnosis of rheumatic fever. He gave a history of repeated joint attacks lasting from one to three weeks

with a sudden onset and considerable pain not relieved by salicylates and with complete recovery between the attacks. He had no carditis, a temperature of one hundred degrees and a white count of thirteen thousand. A routine blood uric acid was nine and six-tenths milligrams per cent and he later developed tophi typical of gout. A second case of gout had no tophi, x-ray changes, or family history, but had recurrent attacks of polyarthritis with complete remissions and a uric acid of five and nine-tenths. He responded to colchicin treatment.

In a brief discussion of gout, Dr. Bauer stressed the recurrent attacks of short duration with complete remissions, without cardiac involvement, usually with a family history, especially in men, often occurring after trauma, sometimes with prodromal symptoms, attacking any joint or joints, very painful, and occasionally associated with recurrent urinary calculi. The younger patients are more apt to have several joints involved and to have more fever.

BACKGROUNDS OF MEDICAL PRACTICE

Humility is appropriate in all thinking persons who must deal professionally or otherwise with problems of human beings, Professor Edward Sapir, head of the department of Anthropology at Yale University, said February 18 in the first of two lectures by him in the series on backgrounds of medical practice. The physician in treating a patient is faced not only by a physical organism but by a tremendously intricate system of habits and customs with roots thousands of years old the speaker said, and his attempts to understand human beings can only be "a series of gropings in a vast night".

There are no such physicians as laymen want doctors to be, the professor further said. The patient frequently looks to the medically trained man for "a scientifically legitimatized papa or mama," who sees all, knows all, and can remove all burdens and worry. Therefore, no one needs more to convey a healthy sense of skepticism than the physician, who realizes that there is "appallingly little" known about why people think, feel, and act as they do, and what relations their behavior has to the physical organism and its well-being. It is entirely possible, according to Dr. Sapir, that mysterious diseases may ultimately be linked up with "cultural sets,"—mannerisms, fancies, preferences.

The pervasiveness of culture, the inheritance of the individual from society past and present, was particularly emphasized by Professor Sapir. Human beings are thoroughly in its grip. No act or gesture can be taken for granted. Ways of walking and talking, even such attributes as appreciation of music, are rooted in instinctive action, but are not in themselves instinctive. The ways of men become accepted merely through use. It is this use alone, and not the nature of the physical organism, which differentiates the people of one nation, one tribe or one age, and those of another. Good will is not sufficient in endeavoring to throw light on the mystery of what man is, Professor Sapir concluded. Of more importance so far as physicians are concerned is "some hunch as to the values of the patient," i.e., some understanding of the culture.

Professor Sapir will deliver his second lecture in this series on March 4 at the Sterling Law building on "Human Beings as Personalities." The series is under the auspices of the Yale University School of Medicine.

FAULKNER HOSPITAL CLINICAL MEETING

The regular clinical meeting was held at the Faulkner Hospital on Thursday afternoon, February 7, at five o'clock.

Two of the cases which had come to autopsy during the past month were discussed.

One of the cases was that of a physician sixty-five years of age who had had injury to his heart due to disease of the coronary arteries about three years ago. This was preceded by moderate angina for a period of several years. After the disturbance which was due to a coronary occlusion the patient had repeated attacks of acute pulmonary edema. These attacks were brought on by disturbance in urination. This disturbance in urination was found to be due to a median lobe of the prostate. Apparently his attempts at emptying the bladder put a severe strain upon the injured myocardium. It was interesting to note that despite the difficulty in urination, by lying down as he urinated he was able to empty his bladder so that there was practically no residual, provided that he did not allow the bladder to fill up beyond six to eight ounces of urine. The heart at postmortem examination showed an extensive degeneration in the wall of the myocardium and only thread-like openings in the coronary arteries. There was a mural thrombus which presumably was the cause of an embolus going to the brain which produced a bilateral hemianopsia and some aphasia. Another interesting feature in the case was the suddenness with which the pulmonary edema could develop. One-half hour before his sudden death the lungs were listened to and only a few râles were heard at the base on one side. At autopsy the lungs were filled throughout with pink frothy material.

The other case was one of adherent pericarditis which had been followed for a number of years, during which time there were several periods of broken compensation. Upon examination of the heart it was obvious that at some time there had been a coronary occlusion with a resulting myocardial damage, but there was nothing in the history to suggest cardiac pain, so that this is another instance of a case in which coronary occlusion occurred without pain. Although this fact has been established before, it is not always appreciated by the medical profession. The other interesting feature of the case was that when decompensation occurred some years ago the hydrothorax was in the left chest rather than, as usually happens, in the right.

Following the presentation of these cases Dr. D. J.

Bristol gave a very interesting and clear-cut description of the best way to handle obstetrical problems. He emphasized the importance of inspection of the cervix as well as palpation of it in the early stages of the pregnancy, in order to make sure that there was no local lesion there. He also emphasized the importance of making sure that the uterus was in proper position so that as it enlarged it did not become incarcerated in the pelvis. He called attention to the extreme importance of having the cervix completely dilated in the first stage of labor and emphasized that this could not be done by manual dilatation. In trying to decide whether labor has actually started, a decision can only be reached by determining whether there are changes in the condition of the cervix. Pains other than true labor pains may simulate the start of labor, but if labor is actually under way the cervix will begin to dilate. Sedatives are important for false labor. The proper use of pituitary extract may help in completely dilating the cervix during the first stage.

He then discussed the indications for applying low forceps and emphasized the fact that the application of high forceps and internal podalic version were major operations. Apparently in recent years the use of high forceps and internal podalic version as a means of delivery have diminished, but he thinks that both of these procedures, especially the latter, still have a definite place in obstetrics.

Dr. H. Bristol Nelson reported on the use of the barbiturates in labor. The preparation which he has employed is pentobarbital (nembutal), and he recommends the initial administration of from 6 to 7½ grains. In the primipara, scopolamine, grains 1/150 can be given in addition. In some cases, if the patient is not asleep, two ounces of ether by rectum may be added. If the labor is prolonged, additional pentobarbital (nembutal) grains 1½ to 3 may be given. In these cases restlessness sometimes develops so that the patient must be watched until recovery from the effect of the drug is complete. He has never seen any permanent, or unfavorable results to either mother or child from this treatment, although in some instances the child is rather slow to begin breathing. He also called attention to the fact that in premature delivery the pediatricians at the present time consider that morphine is a harmful drug for the baby.

The next meeting will be held at the Faulkner Hospital on Thursday afternoon, March 7, at 5:00 P.M. In addition to the usual clinical pathological conference on the cases which have come to autopsy during the month, Dr. Sidney C. Wiggin will present a survey of the complications of anesthesia that have developed at the Faulkner Hospital during the last two years.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Shattuck Street Entrance) Tuesday evening, March 12, at 8:15 P.M.

PROGRAM

Presentation of Cases.

A Trip Around the World by Airplane. By Richard U. Light, M.D., New Haven, Connecticut.

MARSHALL N. FULTON, M.D., *Secretary.*

DR. GARDNER ADDRESSES THE YALE MEDICAL SOCIETY

Dr. Leroy U. Gardner of the Trudeau Foundation at Saranac, New York, addressed the Yale Medical Society, February 13, on the subject of silicosis. He discussed the incidence of this industrial disease and its relation to tuberculosis, emphasizing especially the importance of familiarity with the subject on the part of the physician, because of the prevalence of silicosis in mining industries.

THE MASSACHUSETTS SOCIETY FOR SOCIAL HYGIENE

The Annual Meeting of the Massachusetts Society for Social Hygiene will be held at the University Club in Boston on Tuesday, April 23. Dr. E. Granville Crabtree, President of the Society, has arranged for an address to be given by Dr. John H. Stokes of the University of Pennsylvania. The meeting will be held in the evening. It will be preceded by a dinner meeting in honor of Dr. Stokes at which the election of officers of the Society and other business will be transacted.

The New England Dermatological Society and the Massachusetts Neisserian Society will coöperate with the Society for Social Hygiene in arranging for the reception and meeting for Dr. Stokes.

WILLIAM HARVEY SOCIETY

The next meeting of the William Harvey Society will be held Friday, March 8, in the Auditorium of the Beth Israel Hospital, Boston, at 8:00 P.M.

PROGRAM

Speaker: Dr. Percy S. Pelouze, University of Pennsylvania.

Subject: "Neisseriana."

Chairman: Dr. Harold A. Chamberlin, Professor of Urology, Tufts College Medical School.

Percy S. Pelouze, M.D., Jefferson Medical College, 1902. Assistant Professor of Urology, University of Pennsylvania. Home: Philadelphia.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, MARCH 4, 1935

Monday, March 4—

†4 P.M. Clinic—Medical, Surgical and Orthopedic Services, Children's Hospital and Infants' Hospital (Amphitheatre).

Tuesday, March 5—

1:30 P.M. Radio Program—WEEI. "Typhoid Fever."

†2:30-4 P.M. Ward visit, Massachusetts Eye and Ear Infirmary.

†4-5 P.M. Seminar, Pediatric Laboratory, Massachusetts General Hospital.

4:30 P.M. Radio Program—WBZ. "Tired Legs from Altered Circulation."

Thursday, March 7—

*12 M. Clinico-Pathological Conference. Massachusetts General Hospital.

†12 M. Clinico-Pathological Conference. Children's Hospital.

*3:30 P.M. Medical Clinic. Dr. Christian. Peter Bent Brigham Hospital.

†4:30 P.M. Surgical Clinic. Peter Bent Brigham Hospital.

5 P.M. Faulkner Hospital Clinical Meeting.

Friday, March 8—

5 P.M. Radio Program—WEEI. "Diagnostic Laboratories." "Biological Laboratories."

8 P.M. William Harvey Society. Auditorium, Beth Israel Hospital, Boston.

Saturday, March 9—

*10-12. Medical Staff Rounds. Dr. Christian. Peter Bent Brigham Hospital.

Sunday, March 10—

4 P.M. Harvard University. (Medical School, Building D, Longwood Avenue, Boston.) Free lecture. "Twins and Social Biology." Dr. G. H. Parker.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

February 28—Massachusetts General Hospital, Clinical Meeting, will be held in the Moseley Memorial Building, 8:15-10 P.M.

March 1—Boston University School of Medicine Surgical Clinic at the Boston City Hospital. See page 408.

March 7—Clinic at the Peter Bent Brigham Hospital. See page 408.

March 7—Faulkner Hospital Clinical Meeting. See page 411.

March 8—William Harvey Society. See page 411.

March 11, 12, 13—Surgeons to meet in Jacksonville, Florida (Southeastern Surgical Congress). See page 83, issue of January 10.

March 12—Harvard Medical Society. See page 411.

MASSACHUSETTS DIETETIC ASSOCIATION

March 12—Tuesday, 8 P.M. "The Effect of Diet on Anemia," Dr. Lewis Diamond, Instructor in Medicine, Harvard University Medical School, Associate Physician, Children's Hospital.

March 19—Tuesday, 2 P.M. Field Trip: Visit Storehouse, First National Stores.

April 9—Tuesday, 8 P.M. "Small Hospital Problems," Miss Margaret Copeland, Superintendent, Free Hospital for Women.

March 13—Greater Boston Medical Society Postgraduate Clinic Day at Beth Israel Hospital. Symposium on Diabetes Mellitus, 9:30 A.M.-12:30 P.M. Luncheon, 12:30-1:30 P.M. Symposium on Biliary Tract Diseases, 1:30 P.M.-4:30 P.M. The Annual Dinner-Dance will be held at the Copley-Plaza Hotel. Apply to David B. Stearns, M.D., 485 Commonwealth Avenue, Boston, Mass., for particulars.

April 23—The Massachusetts Society for Social Hygiene. See page 411.

April 29 - May 3, 1935—The American College of Physicians will meet at Philadelphia. For information address Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

June, 1935—Medical Library Association will meet in Rochester, N. Y. For details, address the Secretary: Miss Frances N. A. Whitman, Librarian, Harvard University Schools of Medicine and Public Health, Boston, Mass.

June 27-29 inc.—British National Association for the Prevention of Tuberculosis will be held at Southport, England. Persons desiring further information should write to Miss F. Stickland, Secretary of the Association at Tavistock House North, Tavistock Square, London, W. C. I., England.

July 22-27—Seventh International Congress on Industrial Accidents and Diseases, Brussels, Belgium. The American Committee of the Congress is under the chairmanship of Dr. Fred H. Albee, New York, for the Section on Accidents, and that of Dr. Emery R. Hayhurst, Columbus, Ohio, for Industrial Diseases. The American delegation to the Congress will sail from New York on July 8 and visit London, Amsterdam, The Hague and Paris, and, optionally, Budapest. Physicians interested in the Congress or in the medical tour in conjunction with it, may address the Secretary, Dr. Richard Kovacs, 1100 Park Avenue, New York City.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The Annual Meeting will be held in May. Time, place and subject to be announced.

E. S. BAGNALL, M.D., Secretary.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of March and May at the Weldon Hotel, Greenfield, Mass.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

March 13—Wakefield.
May 8—Winchester.

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Street, Melrose.

MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY

March 7—The meeting will be held at noon in the Hotel Continental, Cambridge, Mass.

NORFOLK DISTRICT MEDICAL SOCIETY

March 26—Fernald School for Feeble-Minded, Waverley. Details to be announced.

May—Annual Meeting. Date, time and place to be announced.

PLYMOUTH DISTRICT MEDICAL SOCIETY

March—Plymouth County Hospital.
April—Lakeville Sanatorium.

SUFFOLK DISTRICT MEDICAL SOCIETY

March 27—Clinical Meeting at the Boston Lying-in Hospital.

April 24—Clinical Meeting at the Children's Hospital.

The medical profession is cordially invited to attend these meetings.

ROBERT L. DeNORMANDIE, M.D., President.
GEORGE P. REYNOLDS, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

March 13—Wednesday evening. The Memorial Hospital, Worcester, Mass. 6:30 P.M.: Buffet supper. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Buffet supper complimentary by the Hospital.

April 10—Wednesday evening. Worcester Hahnemann Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

May 8—Wednesday afternoon and evening. Annual Meeting of the Worcester District Medical Society. The time and place of this meeting will be announced later.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOKS RECEIVED FOR REVIEW

A Summary of the Treatment of Fractures and Dislocations. R. Broomhead. 39 pp. Leeds: Jowett & Sowry, Limited. 3/6d.

Feeding a Family at Low Cost. Statements in this publication have been accepted by the Committee on Foods of the American Medical Association. 16 pp. Illinois: Evaporated Milk Association.

The Crippled and the Disabled. Rehabilitation of the physically handicapped in the United States. Henry H. Kessler. 337 pp. New York: Columbia University Press. $4.00.